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TRANSACTIONS

OF THE

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

FIFTEENTH ANNUAL MEETING

SPOKANE, WASHINGTON

June 6, 7, 1927

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Fifteenth Annual Meeting, Pacific Coast Oto-Ophthalmological Society, Spokane, Wash., June 6-7, 1927

Insert—Carroll Smith, M. D., Spokane, Wash.

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LIST OF OFFICERS

1906— The first attempt to form a Pacific Coast Oto-Ophthalmological Society was interrupted by the San Francisco fire, and nothing was done.

1911— A number of oculists from all parts of the Coast being in San Francisco to attend the Lane Lectures, given at that time by Prof. E. Fuchs, a meeting was called after one of the lectures by Dr. C. Welty and Dr. H. Barkan, and a permanent organization was formed with Dr. J. F. Dickson, Portland, as President; Dr. W. H. Roberts, Pasadena, Vice-President, and Dr. C. F. Welty, San Francisco, Secretary-Treasurer.

1912— A meeting should have been held in San Francisco, but the President, Dr. Dickson, was absent in Europe. The Secretary of the California State Society was to arrange for a meeting in connection with the State Society meeting. It was felt that this arrangement would cause the newly formed Pacific Coast Society to lose its identity and the suggestion was refused. The officers held over until the next year.

1913— First annual—
President, Dr. John F. Dickson, Portland.
First Vice-President, Dr. William H. Roberts.
Secretary-Treasurer, Dr. C. F. Welty, San Francisco.
Meeting held July 1, 2 and 3, 1913.
Guest, Dr. A. E. Davis, New York City.

1914— Second annual meeting was held at Seattle, July 14, 15 and 16, 1914.
President, Clinton T. Cook, Seattle.
First Vice-President, Ed E. Maxey, Boise.
Second Vice-President, John F. Beaumont, Portland, Oregon.
Secretary-Treasurer, C. F. Welty, San Francisco.
Guest, Dr. Robt. H. Elliott, London, England.

1914-1915—President, Hayward G. Thomas, Oakland, California.
First Vice-President, W. K. Seelye, Portland.
Second Vice-President, R. A. Fenton, Portland.
Secretary-Treasurer, Cullen F. Welty, San Francisco.
Third Annual Meeting, San Francisco, June 15, 16 and 17, 1915.

1915-1916—President, Joseph L. McCool, Portland.
First Vice-President, A. R. Irvine, Salt Lake City.
Second Vice-President, George W. McCoy, Los Angeles.
Secretary-Treasurer, Ralph A. Fenton, Portland.
Fourth Annual Meeting, Portland, June 22, 23 and 24, 1916.

1916-1917—President, C. A. Veasey, Spokane.
First Vice-President, P. A. Jordan, San Jose.
Second Vice-President, F. A. Burton, San Diego.
Secretary-Treasurer, R. A. Greene, Spokane.
Fifth Annual Meeting, Spokane, June 18 and 19, 1917.

1917-1918—President, Alexander R. Irvine, Salt Lake City.
First Vice-President, H. B. Graham, San Francisco.
Second Vice-President, R. W. Perry, Seattle.
Secretary-Treasurer, R. R. Hampton and A. J. Ridges, Salt Lake.
Sixth Annual Meeting, Salt Lake City, August 12 and 13, 1918.

1918-1919—President, Cullen F. Welty, San Francisco.
First Vice-President, John J. Kyle, Los Angeles.
Second Vice-President, Fred Stauffer, Salt Lake City.
Secretary-Treasurer, A. R. Green, San Francisco.
Seventh Annual Meeting, San Francisco, August 4, 5 and 6, 1919.

1919-1920—President, Wilson Johnston, Portland.
First Vice-President, Frederick C. Lewitt, San Francisco.
Secretary-Treasurer, Frank B. Kistner, Portland.
Eighth Annual Meeting, Portland, July 29, 30 and 31, 1920.

1920-1921—President, George W. Swift, Seattle.
First Vice-President, E. E. Maxey, Boise.
Second Vice-President, J. O. Chiapella, Chico, California.
Secretary-Treasurer, Ernest C. Wheeler, Tacoma.
Ninth Annual Meeting, Seattle, July 14, 15 and 16, 1921.

1921-1922—President, Fred Stauffer, Salt Lake City.
First Vice-President, P. A. Jordan, San Jose.
Second Vice-President, Henry M. Cunningham, Vancouver.
Secretary-Treasurer, Edw. D. LeCompte, Salt Lake City.
Tenth Annual Meeting, Salt Lake City, September 14, 15 and 16, 1922.

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1922-1923—President, William Humes Roberts, Pasadena.
First Vice-President, Kaspar Pischel, San Francisco.
Second Vice-President, A. W. Morse, Butte.
Secretary-Treasurer, C. Benson Wood, Los Angeles.
Eleventh Annual Meeting, Los Angeles, June 21, 22 and 23, 1923.

1923-1924—President, Frederick A. Kiehle, Portland, Oregon.
First Vice-President, Arthur C. Jones, Boise, Idaho.
Second Vice-President, R. A. Green, Spokane, Wash.
Secretary-Treasurer, C. Benson Wood, Los Angeles, California.
Twelfth Annual Meeting, Portland, Oregon, June 19, 20 and 21, 1924.

1924-1925—President, H. M. Cunningham, Vancouver, B. C.
First Vice-President, C. Benson Wood, Los Angeles, California.
Second Vice-President, Hans Barkan, San Francisco, California.
Secretary-Treasurer, Walter F. Hoffman, Seattle, Washington.

1925-1926—President, Kaspar Pischel, San Francisco, California.
First Vice-President, Glen Campbell, Vancouver, B. C.
Second Vice-President, E. M. Neher, Salt Lake City, Utah.
Secretary-Treasurer, Walter F. Hoffman, Seattle, Washington.

1926-1927—President, Carroll Smith, Spokane, Washington.
First Vice-President, William Mellinger, Santa Barbara, California.
Second Vice-President, Frank Burton, San Diego, California.
Secretary-Treasurer, Walter F. Hoffman, Seattle, Washington.

(The Secretary will be grateful for corrections and additions to the above.)

The Sixteenth Annual Meeting Will be Held in Santa Barbara, California.

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SCIENTIFIC PROGRAM

MONDAY, JUNE 6, 1927

The Fifteenth Annual Meeting of the Pacific Coast Oto-Ophthalmological Society was called to order in the Davenport Hotel at 8:00 A. M. by the President, Dr. Carroll Smith, Spokane, Washington.

Registration

President's Address
Dr. Carroll Smith, Spokane, Wash.

Report of an Unusual Case of Sympathetic Ophthalmia
Dr. R. E. Golden, Walla Walla, Washington
Discussion opened by Dr. H. M. Cunningham, Vancouver, B. C.

A Case of Carcinoma of the Choroid with a Review of Cases Previously Reported by the Writer
Dr. Allen Greenwood, Boston, Massachusetts
Discussion opened by Dr. John E. Weeks, Portland, Oregon

Ocular Deviations, Their Diagnosis and Treatment
Dr. J. A. Smith, Vancouver, B. C.
Discussion opened by Dr. J. Thomas Dowling, Seattle, Washington

Vision in Relation to Industry
Dr. E. M. Neher, Salt Lake City, Utah
Discussion opened by Dr. A. W. Morse, Butte, Montana

MONDAY AFTERNOON

Vitamins and Dietetics in Relation to Oto-Laryngology
Dr. Burt R. Shurly, Detroit, Michigan
Discussion opened by Dr. Frederick A. Keihl, Portland, Oregon

Frontal Sinus Empyema in Young Children With Several Case Reports
Dr. Irving Martin Lupton, Portland, Oregon
Discussion opened by Dr. A. C. Jones, Boise, Idaho

Pyocyanic Stomatitis With Agranulocytic Leukopenia
Dr. F. H. Linthicum, Los Angeles, California
Discussion opened by Dr. Peter A. Jordan, San Jose, California

Business Meeting of the Society

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TUESDAY, JUNE 7, 1927

9:00 A. M.

Trichloracetic Acid in the Treatment of Ulcerative Laryngeal Tuberculosis
Dr. Bertram C. Davies, Los Angeles, California
Discussion opened by Dr. Graham

Further Observations on the Ethmoidal Problem
Dr. Ross Hall Skillern, Philadelphia, Pennsylvania
Discussion opened by Dr. Harris P. Mosher, Boston, Massachusetts

The Reconstruction of a Completely Destroyed Auricle
Dr. Harrington B. Graham, San Francisco, California
Discussion opened by Dr. J. H. Harter, Seattle, Washington

Hypophyseal Tumors in Relation to the Eye and Nose
Dr. James M. Patton, Omaha, Nebraska
Discussion opened by Dr. Geo. W. Swift, Seattle, Washington

TUESDAY AFTERNOON

Binocular Corneal Microscopy
Dr. Lawrence G. Dunlap, Anaconda, Montana
Discussion opened by Dr. James M. Patton, Omaha, Nebraska

Experiences in Extraction of Intraocular Foreign Bodies
Drs. Otto Barkan and Hans Barkan, San Francisco, California
Discussion opened by Dr. Joseph Goldstein, Los Angeles, California

Choked Disc in Intracranial Lesions, the Mechanical Factor in Its Causation
Dr. George W. Swift, Seattle, Washington
Discussion opened by Dr. Walter B. Lancaster, Boston, Massachusetts

Fundus Changes in Cardiovascular-renal Disease
Dr. Walter B. Lancaster, Boston, Massachusetts
Discussion opened by Dr. John E. Weeks, Portland, Oregon

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FIFTEENTH ANNUAL MEETING

OF THE

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY HELD AT SPOKANE, WASHINGTON

June 6, 7, 1927

PRESIDENT'S ADDRESS

CARROLL SMITH, M. D., Spokane, Wash.

According to custom there is first a president's address; the one this year is going to be very brief indeed.

Spokane welcomes you here this year, the first time in ten years. We hope it will not be that long before you come again.

Our society welcomes The American Board for Ophthalmic Examinations and The American Board of Otolaryngology. We greatly appreciate your honoring us by holding your examinations here and participating in our program. The meeting is much more attractive on account of your presence and we hope your trip here will be as pleasant to you as we are pleased to have you.

We greatly regret the fact that our good friend Dr. Ralph Lynch could not come and his absence leaves a big hole in our program. He recently wrote me that on account of the danger that still exists from the Mississippi flood it was impossible for him to leave. I have just received from him a wire which I will read:

> "Please express my sadness and disappointment to all the Fellows and at the same time give them my warmest personal regards, and I sincerely hope that at some future time I may be able to attend one of your meetings in full force and enjoyment. Best wishes for a successful meeting."

I am also exceedingly sorry to announce that two of our most loyal and active members are kept away on account of recent illnesses. Dr. Ernest C. Wheeler recently had an attack of apoplexy. I am glad to announce, though, that his physician informed me a few days ago that he was improving and is now able to be up some. Dr. W. H. Roberts has recently had some heart attacks that made it necessary for him to cancel all engagements and remain quiet for a while. I would suggest that we send telegrams to these three men, expressing our regrets and extending our good wishes.

I take this opportunity of expressing my appreciation for the honor you have conferred on me; I feel that no greater honor can be given one than to be named president of a society of his fellow practitioners.

I also want to thank all who so freely helped me during the year. To the local men and the committees much of the credit for the success of this meeting is due. I mention particularly two names, our very efficient secretary, Walter Hoffman, and my old friend and stand-by Ralph Fenton; they seem to know all about running a society and have helped me more than I can tell you.

Candidates for examinations by the Ophthalmic Board are to report in the Gothic room on this floor at 10:00 o'clock this morning. The clinical and pathological examinations will be held this afternoon at 2:00 o'clock at the Sacred Heart Hospital.

The program will now start. Will each member rising to discuss a paper please first state his name and address so it can be written on the black board, and also come forward so that the stenographer can better hear.

A motion about the telegrams to Drs. Lynch, Wheeler and Roberts will be entertained before I call for the first paper.

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REPORT OF AN UNUSUAL CASE OF SYMPATHETIC OPHTHALMIA

R. E. Golden, M. D., Walla Walla, Wash.

C. H., a farmer, aged fifty-three, accompanied by another person, consulted me March 22, this year. He complained that he noticed that he was commencing to go blind in his good eye two weeks previously. He just thought his vision was blurred and that it would clear up again, but it kept getting worse.

HISTORY

The history obtainable from him was, that sixteen years ago he was splitting wood, when a piece of steel wedge struck his right eye; it was inflamed, but he could not remember it being very painful. But after two or three weeks, he lost all sight in the eye. An eye specialist, who attended him at that time, advised the removal of the eye. His family physician and two or three other general practitioners whom he consulted advised against the removal of the injured eye, even telling him, if he had the eye removed, it would be apt to make the other eye weak.

The patient apparently thought he did not have any further trouble, until about sixteen years later, which was shortly before he consulted me, when as he says, at this time he commenced to go blind in the other eye.

He was a hard patient to get any definite history from. He did not seem to think that there had ever been any pain in his good eye. He did not think it was inflamed any, and he could not remember of having any pain or inflammation in the injured eye for years, or since shortly after it was originally injured. When asked how good his sight had been in his good eye previous to the last two weeks, it was hard to get anything very definite. He said he always attended to his farm work and drove an automobile around town, but he never tried to do much reading for several years past, except large print, and he did not use glasses. Naturally at his age, he would not be expected to do much reading without the aid of glasses.

EXAMINATION

The general appearance of the patient indicated good health. There was no history of infectious or hereditary disease.

Urinalysis showed: Hazy appearance; Sp. gr. 1022. No sugar, no albumen, no Indican, Acetone or Diacetic Acid. Granular casts x.

His vision was, R. E. No light perception. L. E. He could count fingers at three feet. He had comparatively small deep-set eyes. The right eye was not phthisical or atrophied. It was not acutely inflamed or painful, but showed evidence of an old injury. The pupil was immobile and very much contracted, and irregular in outline, there being a small traumatic coloboma of the iris in the lower temporal quadrant. The pupil was about 2 m.m. in diameter. The iris seemed to be bulged out and in contact with the posterior surface of the cornea. It seemed to have a fairly good color. A cataractous lense was lying immediately behind the pupil, anterior chamber obliterated. It looked like a white foreign body lying just inside the pupil. The eye was not soft. Tension seemed to be about normal. There was no light reflex from the fundus, and it was impossible to view the fundus.

The left eye was inflamed slightly, although the patient said it had not been, and was not then painful. The conjunctival and ciliary vessels were congested. The pupil was immobile and about 3 m.m. in diameter and there was very little reaction to a 4% cocaine solution instilled. The ocular tension taken with a Gradle-Schiotz tonometer registered 34 m.m., the normal with this instrument being about 25 m.m. It was not attempted to use atropine to further dilate the pupil at this time. The lense was clear. The vitreous was very cloudy and a view of the fundus was not satisfactorily obtained.

A radiograph of the old injured eye plainly showed that the foreign

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body was still present and that its location was in the vitreous cavity, in the lower nasal quadrant, posterior to the center.

DIAGNOSIS

My diagnosis was a sympathetic ophthalmia, causing the oncoming blindness in his good eye.

As it is a little unusual to have a sympathetic ophthalmia result after so long a period of time has elapsed from the injury of the first eye, unless the first eye has atrophied, or it is in a state of secondary active inflammation, especially if the injured eye has been harboring a piece of steel or copper; and realizing that it was perfectly possible to have an iritis or irido-cyclitis in this man's uninjured eye from other causes than the old injury of the other eye of years ago, it was a little hard to believe that this was a sympathetic irido-cyclitis after so many years of quietness of a possible exciting eye. But taking into consideration that there was an immobile pupil and seclusion of the pupil, which was very little affected by mydriatics and that the vitreous was so cloudy that view of the fundus could not be obtained, it looked reasonable that this was a case of sympathetic iridocyclitis, and I advised the man his blindness coming on was due to the old injured eye, and that it should be removed immediately to try and save what little vision he had left in his good eye. But I did not give him any encouragement that he could get back any of his lost vision.

He was doubtful and very reluctant to have anything done, and I suggested we call consultation. Dr. G. R. Gowen was called, and my diagnosis and advice was confirmed. Still the patient was not satisfied and wanted to "think it over."

We did not see him again for a week, when Dr. Gowen reported that the patient was ready to have the old injured eye removed.

In the meantime the patient had gone to Seattle and said that Dr. Swift had given him the same advice as we did.

By this time the patient could not count fingers but could only see movement of objects before him.

The exciting eye was enucleated on March 30, which was three weeks after the symptoms of blindness had come on in the sympathizing eye. Some other physician whom he had seen, apparently used atropine on the sympathizing eye and the pupil was now more or less free and about 3 m.m. in diameter.

HISTO-PATHOLOGY OF THE ENUCLEATED EYE

The enucleated eye was turned over to a pathologist to have some slides made so they could be shown with the Black-Shields Ophthalmic Lamp, but he did not get satisfactory specimens.

The enucleated eyeball was found to be firm, but slightly irregular in surface contour. There were two small bulging areas of the surface in the outer lower quadrant near the equator. When the eye was bisected from before backwards, there was a gush of aqueous, much more than would be expected to come from the aqueous humor. The vitreous proper was shrunk to less than one-half, and instead of being of a gelatinous consistency, it was more of a fibrinous nature. It was surrounded by the large amount of aqueous which apparently kept the eyeball filled out retaining its shape and tension.

The lense maintained its form but looked like a piece of white chalk and was very friable. It occupied a position in the anterior chamber, lying against the posterior surface of the iris, bulging the iris out against the posterior surface of the cornea, and the suspensory ligament was entirely gone as the lense dropped out when the eye was opened.

The foreign body was lying in the lower inner quadrant, firmly encapsulated in a mass of exudate in the vitreous. Upon close inspection and under a magnifying glass, the foreign body looked as though it might have been a little piece of rock two or three millimeters in diameter. Its surface

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showed a certain amount of disintegration, but as it was easily picked up with a magnet and after a few days showed rust stains, it was decided that the foreign body was a piece of steel as the history of the case suggested.

AFTER-TREATMENT

After the operation, the patient was given sodium-salicylate, 15 grs. intravenously, daily for four days, and also 15 grs. orally three times daily. Then the sodium-salicylate was continued by mouth, 20 grs. three times daily for three weeks more. He was also given, beginning the day after the operation, six doses of lacto-albumen, 5 c.c. daily, intra-muscular injections.

The inflammation in the sympathizing eye subsided soon after the removal of the irritating eye, and in a few days his vision commenced to improve, and four weeks after the operation he could get around by himself and thinks he can recognize his friends.

COMMENTS

Regarding sympathetic ophthalmia, some authorities claim that it is useless or too late to accomplish any good in saving vision, to enucleate the irritating eye after symptoms of inflammation are well established in the sympathizing eye. In our case the inflammation was so well established in the sympathizing eye that the vision was reduced to observation of movements of large objects; yet after the removal of the irritating eye, the vision later became considerably improved.

Also according to different authors the following facts are pertinent:

Fuch says[1]: "It is only in rare instances that a foreign body is tolerated for any length of time in the eye without setting up inflammation, the body itself either remaining free or becoming encapsulated in an organized exudate."

Again from Roemer[2]: "Both experiments and much clinical experience have proved that there is scarcely a single kind of foreign body which can be borne in the eye without harm."

Also Roemer says[3]: "That the average time of interval between injury and onset of sympathetic irido-cyclitis is six to ten weeks."

And also we find: "The longer the time that has elapsed since the injury, the more doubtful is the diagnosis and the more careful must other forms of iritis be excluded."

Roemer also says[4]: "A fibrinous irido-cyclitis alone is able to excite a sympathetic inflammation in the other eye." Rare exceptions are noted where sympathetic inflammation may follow a purulent inflammation of the uveal tract, but the purulent may pass into a fibrinous inflammation. It is, however, beyond doubt that the puss agents are not the agents of sympathetic irido-cyclitis; therefore, if a sympathetic irido-cyclitis follows a purulent infection, it is supposed there was a mixed infection present.

In the pathogenesis of sympathetic ophthalmis, there are several theories advocated, among which may be mentioned The Leber-Deutschmann's Migration Theory[5]. The Ciliaeural Theory of Schmidt-Rimpley[6], and the Metastasis Theory as suggested by Mackenzie and elucidated by Berlin[7].

According to Leber's experiments in animals, the results of which agree in general with observations upon man; the tolerance with which foreign bodies are borne in the eye, depends upon the following conditions: First, whether the foreign body that enters the eye is aseptic or not. Second, upon its chemical character. Third, the location in the eye where the foreign body is imbedded.

Foreign bodies like fragments of glass, if entering the eye in an aseptic condition, are much more likely to be tolerated than a chip of steel or copper, which in time usually dissolves from chemical action in the eye. And as to location: Foreign bodies imbedded in the lense are less apt to produce trouble (aside from the cataract produced), and are more readily tolerated than in any other intra-ocular location. The foreign body is more apt to produce trouble if in the ciliary region or vitreous.

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However, the prevailing opinion among authorities seems to be that the infection in sympathetic-ophthalmia is due to a micro-organism which has not yet been isolated; and due to a pathogenic agent which has a predilection for exciting inflammation in the eye.

CONCLUSION

The excuse for reporting this case is on account of some of the unusual features connected therewith:

A piece of steel being tolerated in the vitreous, and the long interval of quietness in which it remained in the exciting eye.

The steel being encapsulated without any apparent chemical action taking place in the eye.

The injured eye retaining its form and fullness without any apparent shrinkage.

And, an improvement and return of a considerable amount of vision in the almost blind sympathizing eye after removal of the irritating eye.

BIBLIOGRAPHY

1. Fuch's Text-book of Ophthalmology, 4th Ed., page 330.
2. Text-book of Ophthalmology, Roemer, page 372.
3. Text-book of Ophthalmology, Roemer, page 357.
4. Text-book of Ophthalmology, Roemer, page 355.
5. Text-book of Ophthalmology, Roemer, page 363.
6. Text-book of Ophthalmology, Roemer, page 366.
7. Text-book of Ophthalmology, Roemer, page 369.

Discussion opened by Dr. H. M. Cunningham, Vancouver, B. C.:

This honor is purely accidental. I was responsible for the man who should have led this discussion, and since he found it impossible at the last moment to do so I had to fill the breach. I do very little eye work and have not kept up on recent ophthalmological literature, so that I do not feel qualified to really discuss this paper, but may be able to start something, and should not be surprised to have it one of the best discussions of the morning. I see men all over the room watching their chance to jump in.

There are, to my mind, at least five interesting features to this case.

In the first place—Is the writer justified in claiming that a lapse of sixteen years is unusual?

Second. Was this really a case of true Sympathetic Ophthalmia?

Third. What effect, if any, did the removal of the injured eye have on the progress of the case?

Fourth. Is the writer justified in presuming that this case is finished?

Fifth. The unsolved problem of Sympathetic Ophthalmia and the best method of treatment.

Now most of you have seen or had cases of sympathetic ophthalmia, and instead of quoting cases indiscriminately, I think we will have a more valuable discussion if the quoting of cases, together with the discussion, be definitely aimed at one or more of the pionts suggested.

Dr. John E. Weeks, Portland, Ore.:

Mr. President and Gentlemen: In the N. Y. Eye and Ear Infirmary Reports of 1894, I reported an unusual case of sympathetic ophthalmia which occurred forty-two years after injury to the exciting eye. The patient was observed by a number of the surgeons at the Infirmary all of whom concurred in the diagnosis. I have never seen a case of sympathetic ophthalmia occurring long after the injury to the exciting eye took place that did not give a history of periods of excitation within two months of the beginning of the sympathetic disturbance.

In the case just reported there was no history of irritation of the exciting eye before the sympathetic inflammation began. This fact would lead me to doubt the correctness of the diagnosis, although I am far from stating that it was not a case of sympathetic ophthalmia. The affect of removing

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the exciting eye after sympathetic inflammation has not been encouraging in my experience. The affection of the sympathizing eye has developed, apparently without much if any abatement after the exciting eye has been removed. In a number of cases sympathetic ophthalmia has developed some time after the exciting eye has been removed. In one case three weeks elapsed after the exciting eye was removed before the sympathetic inflammation began and then a typical case developed.

The reader of the paper stated that a purulent inflammation affecting one eye was not apt to excite sympathy in the fellow eye. I have no doubt that many of those present have seen a metastatic chorioiditis following a meningitis or a pneumonia, particularly in young individuals. It has never been my experience to see sympathetic ophthalmia as a result of such an infection. The eye may become shrunken and decidedly atrophic, but sympathetic ophthalmia does not develop. Removal of an eye so affected need not be urged because of danger of sympathy in the fellow eye.

Dr. Joseph Goldstein, Los Angeles:

The case under discussion has many points of importance which should be cleared up before the diagnosis of sympathetic ophthalmia can be made.

Permit me, before entering into this discussion, to state, that in a fairly busy practice extending over several years, our office has not made a diagnosis of this condition. The two senior eye surgeons of the Los Angeles General Hospital, who have been in service for more than twenty years, have not recorded such a diagnosis. One stated that in the years gone by, he believed he had seen one case.

The symptomatology and findings noted in this special case have been observed in our practice many times. This condition has been observed with no exciting eye etiology.

The report does not state that sinus infection was not present; that apical infections were absent; that the tonsils were absolutely negative, and other foci of infection eliminated. Furthermore, no serological examinations were made and no venereal history eliminated. These and many other causes could produce this picture and until all these are eliminated, I cannot agree with the writer.

I should be very happy to hear opinions of older men on this important subject.

Dr. Patton, Omaha:

I wish to congratulate the author on the excellent outcome of his case. To me it seems like a definite case of sympathetic ophthalmia. I agree with Dr. Weeks in that I do not recall a case of sympathetic ophthalmia that was not preceded by a period of irritation.

I would like very briefly to report a case I have had under observation for the last seven months. The patient, a boy of fifteen, was injured about a year previously. There was a ten-day period of irritation before consulting us, but on presenting himself, the clinical picture of sympathetic ophthalmia was complete. Immediate enucleation of the injured eye, vigorous administration of sodium salicylate intravenously and by mouth, mercurial inunctions and foreign protein, atropine and heat locally, resulted in reduced congestion, a dilated pupil, and in ten days a vision of 20/50. In spite of the improvement, he was subject to relapses, periods of irritation, low fever and finally secondary glaucoma requiring operation. His eye has been quiet now for over a month with normal tension and a vision of 20/30 or better, but it is too soon to even hope that there will be no more flareups. Treatment must be continued for several weeks or even months after all evidence of irritation has subsided.

Dr. A. C. Jones, Boise:

I wish to report a case of a girl of 16 years of age. Twelve years after being injured with one of these giant caps she developed sympathetic ophthal-

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mia. I saw her while I was practicing in Butte, Montana. Dr. Petter Potter saw this case in consultation with me.

The girl was confined in the juvenile ward of the county jail. She was brought in by the sheriff. Four days previous to my examination, she began to have photophobia in her seeing eye and vision had diminished rapidly and photophobia had increased. Vision in left or exciting eye nil. It showed corneal scars, prolapsed adherent iris and minus tension. Right eye vision 20/100 marked circumcorneal and conjunctival irritation and many deposits on Decments membrane. The exciting eye was X-rayed and enucleated. Four small pieces of copper were found in the eye. Salvarsan was given at five-day intervals for ten doses, along with massive doses of sod-salicylate. Her vision improved rapidly after the second injection. The photophobia decreased. At the end of four weeks her vision in the right eye was 20/40 and in seven weeks it was 20/20-3. She had no further trouble.

Dr. R. L. Nourse, Boise:

In order to enlarge the discussion somewhat—particularly with regard to the pathology and diagnosis—I would like some of those who discuss this paper to tell us how early we may get sympathetic ophthalmia following a penetrating wound of the eye.

We have heard in the case just reported that it may occur fifteen to twenty years after the injury. I would like to know how soon after the injury it may occur. I had a case which developed within four days to the extent that vision in the sympathizing eye had so failed from Friday, the date of injury, by a small sliver of steel penetrating the injured eye, to the Monday following, the patient was unable to distinguish his wife from another woman across the room; although vision prior to the injury had been normal.

The injured eye was enucleated on the fifth day and it was three weeks before vision in the sympathizing eye returned to normal.

I diagnosed it as sympathetic ophthalmia. If I was wrong will some one tell me what it was. If I was correct how would the theory of infection account for the rapidity of the onset.

Dr. Wm. D. Donoher, Salt Lake City:

I agree with Dr. Golden in his diagnosis for the reason that I don't think it is anything else. If he failed in his diagnosis, somebody must suggest some other thing that might be the cause of the trouble. As to the administration of Salvarsan, I don't believe there is any affection of the eye not benefited by Salvarsan, but . . . If he was benefited by this administration, I don't believe this proves it was not sympathetic ophthalmia. I do believe this though. That the longer the foreign body has remained in the eye the less violent the affection the more time one is given to make a diagnosis and instituting his treatment.

I think further that the removal of a foreign body will give a sense of security. I think many, many times sympathetic ophthalmia will develop after removal of foreign body, and . . .

As to seeing many, many cases of sympathetic ophthalmia, I think most men who have seen one, have seen quite enough. They don't hunt for others.

Dr. J. F. Dickson, Portland:

I don't want to say very much, as I think Dr. Golden has stated the case very well, and congratulate him on the happy result of his treatment. Whether sympathetic ophthalmia or not is a question. I am inclined to think it is, but to my mind it emphasizes one thing, and that is, with a blind exciting eye, and the good eye showing well marked symptoms of irido-cyclitis why not enucleate the exciting eye and so get rid of a potential menace to the good eye. If the case were sympathetic ophthalmia it should help the good eye, and if not it would remove a source of irritation to it that would be beneficial. I am old fashioned, I know, but I never have hesitated to advise removal of a painful blind eye when the other was having recurrent

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attacks of inflammation. This is my rule, whether the good eye has real sympathetic ophthalmia or only attacks at intervals of sympathetic irritation. The blind eye is a menace in any case that should not be allowed to exist.

Dr. Hamilton Stillson, Seattle:

You remember that a young man asked Socrates whether he should marry the young lady of his choice and Socrates replied: "Why ask?"

Cunningham is a fine chap to start the discussion, but why ask is this a case of sympathetic ophthalmia? You are going to treat it just the same. And is the recovery the final result? Well, that of course is for Allah to determine. . . . Of course, you are going to enucleate to be on the safe side and so in the words of Socrates, again, "Why ask?"

Dr. Walter B. Lancaster, Boston:

My personal experience with sympathetic ophthalmia is very limited. It is very instructive to compare the number of cases of sympathetic ophthalmia in the World War with previous wars. Although the number of eye injuries in the recent conflict was extremely large, blindness from sympathetic ophthalmia was a great rarity. This was unquestionably due to the better treatment, and is conclusive proof of the importance of proper treatment. May we not say that the age of sympathetic ophthalmia is somewhat passed? It is rather a bad sign if a man sees many cases in his own practice. My own feeling is that sympathetic ophthalmia is such a disastrous occurrence that it is far better to give the patient the benefit of the doubt, and remove the eye.

I may cite two cases of my own. In the first an eye was enucleated following an operation, not because sympathetic was feared or suspected, but because it was a painful blind eye. The eye was sent to the laboratory, and in a few days the pathologist came with the report that the eye showed unmistakable signs of the type of inflammation known as sympathetic ophthalmia. The other eye did perfectly well. The disease was not in a virulent form. This illustrates the fact that while it is not possible to make a positive diagnosis clinically, it is possible microscopically. Some day we shall understand the pathology as well as we do the treatment.

The other case I might mention is a doubtful one, since there was no occasion for microscopical examination. Following an operation of iridencleisis, both eyes showed a mild uveitis with well marked deposits on the endothelium of the cornea and slight ciliary injection and reduced vision. Now we have always heard that injuries (including operations) to the eyes involving the iris or ciliary body are liable to be followed by sympathetic; and yet thousands of iris-inclusion operations for glaucoma have been done with very few cases of sympathetic reported. This case happened to be seen by Dr. Wheeler of New York, and he said, "What else can it be?" No focus of infection could be found, and the man was in exceptionally good physical condition. The treatment included vigorous use of salicylates—it was before the days of diphtheria antitoxin treatment of sympathetic ophthalmia—recovery was complete. At the present time sympathetic ophthalmia in Boston is being treated with large doses of horse serum (diphtheria antitoxin), and Dr. Verhoeff who sponsors this treatment is very enthusiastic about it and feels that it is a specific.

Dr. Allen Greenwood, Boston:

I do not think I can add much to the discussion or that part I have heard. I think Dr. Dixon hit the nail on the head and Dr. Lancaster followed him up.

During the late war, I had a chance to be in France for nearly two years and during that time I only saw one case of sympathetic ophthalmia. The reason they did not have sympathetic ophthalmia in the late war was due to the fact that men with badly injured eyes had them removed; so I think the whole substance of this discussion is to call attention to the advisability of removing a blind, painful eye that has been blinded from injury, whether

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received during some operation or the result of some penetrating injury. I do feel this, however, in regard to sympathetic ophthalmia, that I doubt if true sympathetic ophthalmia very frequently follows unless the eye injured was in a condition of irido-cyclitis. I received a letter some time ago asking my position. Did I think that sympathetic ophthalmia would arise after the formerly injured eye was white and quiet. I wrote back that I did not believe true sympathetic ophthalmia could.

I think if Dr. Golden could have had the injured eye sent to Dr. Verhoeff, the question of sympathetic ophthalmia could have been settled immediately. I am sure cases are reported as sympathetic ophthalmia when the condition is not the result of a previously injured eye, but comes from some of the causes causing uveitis.

Dr. Kaspar Pischel, San Francisco:

As Dr. Golden had difficulty in procuring good slides, I would like to draw your attention to this year's report of the Pathological Committee of the Section on Ophthalmology of the American Medical Association. (Presessional Report, page 305.)

"Through the industry of Major Callender, curator of the Army Medical Museum, an excellent collection of specimens, slides, photographs, etc., related to the pathology of the eye is gradually being built up, and it is hoped that this collection will in time be available not only for teaching purposes, but also for special research and investigation. To those who send in specimens for diagnosis, a full report is given, together with a slide and, if requested, a copy of any photographs that have been taken."

Dr. Wm. Lord Blake, San Francisco:

Mr. President, I should like to suggest that the Chairman of this meeting appoint some one of our number who is particularly interested in the pathology of the eye to submit to this meeting next year a paper on the pathology of sympathetic ophthalmia. I am sure that those of us who are so unfortunate as to be obliged to remove an eye because of sympathetic manifestations will be very glad to put their specimens in his hands for pathological examination. I make this suggestion because it is apparent from the remarks made here this morning that we are not all in agreement on the pathology of sympathetic ophthalmia.

Dr. R. E. Golden, Walla Walla: (Conclusion)

When I had the audacity to present anything before this body, I felt like a lamb being brought up for slaughter. I expected it to be unmercifully slaughtered, which it has been, and I appreciate I brought up an interesting subject and it has been thoroughly discussed, and we have learned a lot from it, and it has done me a lot of good. As far as any particular questions having been asked, I think they have most of them been answered.

I would like to congratulate Dr. Goldstein on his never having had any sympathetic ophthalmia patients.

Dr. Nourse asked about the diagnosis of his case. I am not prepared to answer that and we would be glad to have someone help us in ours. I am pleased to know that there are really cases of sympathetic ophthalmia seen by a great many men. I thank you.

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A CASE OF CARCINOMA OF THE CHOROID WITH A REVIEW OF CASES PREVIOUSLY REPORTED BY THE WRITER

ALLEN GREENWOOD, M.D., Boston, Mass.

Into our special field of ophthalmology there occasionally come conditions so rare that the average ophthalmic surgeon never, during a busy life of hospital and private practice, sees them. One of the rarest of these is carcinoma of the choroid which is so unusual that up to a few years ago only a score or so of cases had been reported. One of our leading consulting ophthalmic surgeons, after a life long experience in hospital and private practice, reports never to have seen this condition and one might naturally feel that a disease so rarely seen was of very minor importance. If, however, one ophthalmologist sees a group of such cases it would seem wise that his observations be recorded especially in view of the known fact that carcinoma is yearly becoming more common and constantly exciting a wider interest in its manifestations, its causes and treatment.

The writer has, in the past, reported four cases and now adds a fifth to the group.

Miss B. A. F., aged 55, came to my office November 30, 1926, complaining that she did not see as well as formerly and that recently the left eye had had blurred vision. She was a very robust appearing woman with good color and looking younger than her stated age. Right eye vision= 20/20. Left eye, vision = 20/200. Right eye, field of vision normal. Left eye, field of vision quadrant scotoma up and in, reaching to within 10° of the center. Right eye, in the extreme lower part of the fundus the retina was slightly elevated (1 to 2 dioptres), otherwise normal. Left eye, nearly the whole lower half of the retina separated almost up to the disk and macula. Three disk diameters down and out from the disk was the point of greatest elevation (7 dioptres) and here beneath the retina could be seen a round white discoid mass of 3 or 4 d.d. in size showing less white away from its center. The retinal vessels over this area were enlarged and more tortuous. Tension with Schiotz tenometer 24 m.m. o.d. Transillumination not appreciably affected. The appearances were so suggestive of the carcinomas of the choroid previously seen that further inquiry into the patient's past history was pursued with the information that tumor of the breast had been removed two years previously and that she was having occasional X-ray treatments to prevent its recurrence. The patient's family physician was notified of the findings and told that they indicated a general carcinosis from a local recurrence penetrating into the lung.

December 14, 1926, patient seen again and vision found much worse with the retinal separation more extensive and the white appearance of the mass beneath much less marked. Still looked in fine physical condition. Pilocarpine ordered to prevent possible glaucoma. December 23, 1926, not much change. Seen in consultation by one of our leading American ophthalmic surgeons who agreed with the findings in both eyes, but questioned the diagnosis of metastatic carcinoma in a patient in such apparently good health. At this time, a small swelling appeared on the frontal bone in the mid line. December 31, 1926, not much change. January 6, 1927, came in with a marked swelling of the left eyelids, a proptosis of 4 or 5 m.m. and a slight chemosis. Tension of eye not increased. Diagnosis of acute tenonitis from fresh carcinoma emboli in the orbit. Under ice compresses this inflammatory condition disappeared in five days. January 31, 1927, left eye, retina wholly separated and eye nearly blind. Lids normal, but the proptosis has returned indicating a growing carcinoma of the orbit. During the month of February patient lost flesh very rapidly and symptoms of abdominal disturbances appeared. In bed most of the time. March 1, 1927, left eye wholly blind and markedly proptosed. Pupil small from the pilocarpine. No inflammation.

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Right eye, separation of retina in upper quadrant with a whitish sheen from beneath. Lower half of retina separated. Vision much reduced and patient in a drowsy condition and somewhat incoherent from evident metastatic brain involvement. Soon after patient became comatose and died March 4, 1927.

Ophthalmic diagnosis:

Bilateral metastatic
Carcinoma of Choroid
Carcinoma of left orbit
Death from general carcinosis.

A reference to my four cases previously published shows that two of them came under observation when they were apparently in good condition and the first suspicion of trouble of a serious nature elsewhere came from visual disturbances and the observation that a tumor was developing in the eye. The present case was of this type and thus three of the five cases which I have observed teach us not to be deceived by the healthy appearances of patients who show a white flat discoid mass under the retina with a rapidly spreading separation of the retina, especially if they give a history of a previous operation for carcinoma. Secondary glaucoma, however, can be cured or prevented in most cases by the use of miotics, though this complication is much less likely than in choroidal sarcomata.

For the differential diagnosis of carcinoma and sarcoma the following table previously published is helpful:

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DIFFERENTIAL DIAGNOSIS

	Metastatic Carcinoma	*Sarcoma*
Situation	Posteriorly, usually temporarilly and not far from the macula.	Usually equatorially, or farther forward, rarely near disk.
Shape	Flat discoid with thin edges—rarely elevated more than two or three m.m.	Rounded, nodulated.
Color	Gray or white, without pigment.	Usually dark gray or showing much pigment and mottled.
Projection	Very little if any projection into the vitreous.	Projecting far into the vitreous early.
Growth	Spreads rapidly in all directions and ultimately surrounds the disk, but always flat.	As size increases slowly projects more and more into vitreous.
New vessel formation	Not present.	Usually present.
Retina	Very slightly projected at first by the tumor growth and with little subretinal fluid—separation of the retina around the tumor comes quickly and spreads rapidly as the tumor grows and soon becomes complete.	Projected into the vitreous early by the tumor and separation of the retina around the tumor comes slowly and does not become complete until much later.
Vision	Early disturbed and soon lost owing to rapid growth under or toward the macula.	Vision centrally not early disturbed and not quickly lost unless glaucoma appears—eyes with sarcoma often enucleated with vision normal.
Tension	Not usually increased in early stage and usually amenable to miotics.	Usually raised early and acute glaucoma almost inevitable if eye not enucleated early enough — not affected favorably by miotics.
Enucleation	Almost never necessary and of no value to the patient unless for otherwise unrelieved pain—does not prevent metastases.	Always necessary as early as possible and of great value to the patient in preventing metastases.
Occurrence	Always metastatic and primary focus or other metastases can usually be found—often bilateral.	Always primary—seldom, if ever bilateral and causes metastases later.
Treatment	Palliative.	Early enucleation.
Prognosis	Inevitably bad.	Usually good, if seen early, though metastases of the liver may occur years later.

A study of my cases discloses several facts worthy of mention. In all five cases either an autopsy, an X-ray or physical signs showed involvement of the lungs. With the lungs involved, it is easy to understand how cancer emboli can be taken up by the pulmonary vein, carried to the heart and thence through the arteries to all parts of the body.

The presence of cancer emboli in the arterial circulation is well illustrated by a case of Ishihara, who described a primary mammary carcinoma with multiple carcinomatous emboli in the choroidal capillaries. There were

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three isolated tumor nodules in the choroid and eleven distinct capillary emboli composed of carcinoma cells. In one of Week's cases an eye with a choroidal tumor was removed and the growth found to be carcinomatous with the primary source in the lungs only discovered at autopsy. A great many of the cases tabulated by Suker and Grosvenor showed an involvement of lungs when the records indicated a thorough general post-mortem examination.

The author queries as to how often, if ever, a choroidal carcinoma occurs until after some involvement of the lungs.

Pain and hypertension are the only complications which, if unrelieved by treatment, call for an enucleation of the eye, unless one is in doubt as to the differential diagnosis between metastatic carcinoma and sarcoma and, in such a case, the removal should only be advised after an exhaustive physical and X-ray examination has proved negative as to carcinoma.

Discussion opened by Dr. John E. Weeks, Portland, Ore.:

Because of the development of the choroid from mesoblast, primary carcinoma, an epiblastic tissue, is not possible in that membrane. We see carcinoma in the choroid only as a metastatic growth. As Dr. Greenwood has stated, metastatic carcinoma in the eye is seldom observed, largely because it is rare, but to some degree because the duration of life after the onset of this complication is seldom more than six months and often much less, permitting some of the cases, at least, to escape observation. Oatman (Am. Jour. of the Med. Sciences, Mar., 1903) collected the reports of thirty cases in the literature and added one of his own. Sukor and Grosvenor (Ophthalmology, 1913) reported a case and found sixty-four cases recorded in the literature.

Metastatic carcinoma of the choroid is usually a part of a general carcinosis. The short ciliary arteries appear to be the avenues through which the carcinomatous emboli reach the choroid. When the ciliary body is involved, it is usually due to extension from the choroid; however, there are two cases of metastatic carcinoma developing primarily in the ciliary body reported and one of development in the iris, the carcinomatous emboli evidently reaching these parts through the long ciliary arteries. Primary carcinoma of the ciliary body and of the iris is possible as it may develop from the cells of the pars ciliaris and pars iridis retinae. The writer has examined microscopically two such growths that developed from the ciliary body.

In the writer's experience five cases of metastatic carcinoma of the choroid have come under observation, two of which have come to section. In one case the new growth, at its thickest portion, measured three millimeters, was flat and extended over a relatively large area posteriorly and temporally. A similar growth also appeared in the fellow eye. The patient, aged 56 years, gave a history of tumor of the breast, removed by amputation, three years previously. The patient died three months later. In one case the tumor measured 9 m.m. in thickness, was convex anteriorly, and although it passed imperceptably into normal choroid it did not extend over a large area. It was situated to the temporal side and a little above the macula. The diagnosis was choroidal neoplasm and enucleation was advised. The patient, 49 years of age, appeared to be in excellent health and was so reported to be by her family physician. I was of the opinion that the growth was in all probability a "flat" sarcoma. On microscopical examination the growth appeared to be adeno-carcinoma and was so reported. The patient died three months later of carcinoma of the lung. This occurred in 1903. Of the cases observed by the writer, the primary growth was situated in the breast in three, lungs one, liver (?) one.

Pathology—The carcinomatous cells that invade the choroid are of glandular epithelial type; they are arranged in irregular alveoli, of which the connective tissue trabiculae may be very delicate or may be thick and dense. The alveoli may be large and the cells arranged in single or double rows on the connective tissue frame work presenting bead-like prosses here and there, the centers of the alveoli being filled with fluid. The arrangement causes the

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growth to present the appearance of adenoma, with which it has been confounded (Gayet). The retina and optic nerve may be secondarily involved, deposits of carcinoma cells appearing in them. The vascular supply is small. The normal choroidal vessels are crowded to the periphery and may present a congested condition at the margins of the growth. There is a scant accumulation of small cells in the tissues of the choroid in the vicinity of the growth.

The table to aid in the differential diagnosis between metastatic carcinoma and sarcoma, as presented by Dr. Greenwood, is on the whole very good. Sometimes the projection into the vitreous is considerable, approaching that of flat sarcoma. It has been the fortune of the writer to have seen four, possibly five, cases of sarcoma of the choroid, beginning at the macula. We must not forget that sarcoma of the choroid is sometimes metastatic. Two well authenticated cases are on record, one by Meigs and deSchweinitz (Am. Jour. of the Med. Sciences, Vol. 28, No. 2, page 193), and one by Weiner (Trans. Sec. on Ophth. A. M. A., 1902).

In regard to treatment I am fully in accord with Dr. Greenwood, it can only be paliative.

Dr. A. W. Morse, Butte, Montana:

I would like to report a case which is suggestive to me of this condition, although I did not make the diagnosis.

A patient who had had a breast amputation for carcinoma at the Mayo Clinic three years previously, and whom I had refracted, came to the office because of the failing of vision in the left eye, the vision in the right eye being quite low. A +1.00 added to the old correction gave the same vision as she had before. I was unable to find any pathology in the eye. Two weeks later she came in again complaining. By giving her +4.00 more, or a total of +5.00, behind her old original correction, she had almost normal vision. The retina was raised as in beginning detachment, but I did not make a diagnosis as to what was causing the trouble. I considered a new growth, but thought the change was too rapid, and the vision too good for that condition.

About that time she returned to the Mayo Clinic because of other disturbances, and became totally blind within two weeks, or four weeks from the time she was first seen. I did not have an opportunity to examine the eye again. Thirty-nine days from the time she had the eye disturbance she died of secondary carcinoma of the brain. There were also secondary foci in the lungs.

Dr. Greenwood (Conclusion):

I want to thank Dr. Weeks for his very illuminating and kind discussion on the subject of my paper and my object has been attained if it enables Dr. Morse and others to recognize carcinoma when found and make the diagnosis early.

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OCULAR DEVIATIONS THEIR DIAGNOSIS AND TREATMENT

J. A. SMITH, M. D., Vancouver, B. C.

It is the object of this paper to present in a concise and systematic form the different muscular anomalies which commonly give rise to asthenopic symptoms, with suggestions as to their treatment—non-operative and operative—illustrated here and there with reports of interesting cases.

In my opinion, the study of the different forms of ocular deviation with their proper diagnosis and treatment, constitutes one of the most interesting branches of present-day ophthalmology.

Under the heading of Ocular Deviations can be classed anything, from the non-apparent "phorias" to the well-marked "tropias," and more and more, as the days go by, we see marked asthenopic symptoms as vertigo, headache and nausea, overcome by a reasonable diagnosis and treatment. In early cases, sight can be preserved by treatment, and later on, even if it is impossible to improve the vision, much can be done from a cosmetic standpoint. In this latter regard, age and sex do not seem to constitute such an important factor as one might suppose—two of my recent patients were men over sixty years of age—who had had divergent squints for years. Both were extremely grateful and quite proud of their post-operative appearances. After some consideration, it seems to me the most practical classification would be as follows:

1. Cases of Convergence Insufficiency.
2. Cases of Convergence Excess.
3. Cases of Divergence Insufficiency.
4. Cases of Divergence Excess.
5. Cases of Convergence Excess with a Divergence Insufficiency.
6. Cases of Divergence Excess with a Convergence Insufficiency.
7. The vertical deviations.
8. Cases of paralysis of one or more ocular muscles, either complete or partial.

The first four named are generally spoken of as the latent forms or "phorias"—*Esophoria* and *Exophoria*. The next two are generally manifest and spoken of as "tropias"—*Esotropia* and *Exotropia*, and are comitant.

The vertical deviations may be both latent and manifest, spoken of as *Hypertropia* and *Hyperphoria;* also comitant.

Paralytic deviations are non-comitant, the deviation increasing as we carry the vision into the field of the paralyzed muscle.

1. *Cases of Convergence Insufficiency.*

Convergence Insufficiency may be:

(a) Accommodative.

(b) Non-Accommodative.

The patient complains of drawing pains and headache after reading or engaging in close work for a time. All tests, as Screen, and Parallax, Maddox Rod and Phorometer, show an exophoria for distance which is more marked for near. The convergence near point is remote. Prism convergence is low, while prism divergence is normal.

In the treatment of Convergence Insufficiency, all errors of refraction should be corrected. More satisfactory results can be expected if the patient is myopic. In the case of *Hypermetropia,* a difference of opinion exists—some holding that the prescribing of convex lenses tends to aggravate the condition. Personally, I think it would be wise to compromise by reducing the strength of the lenses somewhat more than we generally do in uncomplicated cases. Prism exercises with prisms base out, if persisted in tend to increase the power of convergence, and is to be recommended as the best non-surgical treatment. Prisms placed base in, tend to overcome some of the difficulty for near work, but prisms placed horizontally for wear, are not to

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be recommended. A cure can generally be obtained in these cases by the advancement of one or both Interni. One eye only should be done at the first sitting and the result noted. If necessary, the effect can be increased by the advancement of the Internus of the other eye. Local anaesthesia is best.

In the Non-Accommodative type, the treatment should be directed along general Hygienic lines. Tonics should be given if indicated, and the patient's general mode of living corrected. The use of alcohol and tobacco should be investigated.

2. *Cases of Convergence Excess.*

This form of Esophoria is practically always associated with uncorrected hypermetropia, hence the symptoms are those of this condition. All the tests, as Screen and Parallax, Maddox Rod and Phorometer, show Orthophoria or slight degree of Esophoria for distance, increasing for near—Convergence near point, normal or abnormally close—Prism Convergence and Prism Divergence, normal. The treatment consists in fully correcting the Hypermetropia.

3. *Cases of Divergence Insufficiency.*

This form of Esophoria, although comparatively rare, may cause very severe asthenopic symptoms which do not seem to be inaugurated by near work.

For distance, all tests previously mentioned show marked Esophoria, which is decidedly less for near. The Convergence near point is normal. Prism Convergence is normal. Prism Divergence is low; less than 2 or 3°.

This form of Esophoria is very amenable to prisms placed base out for constant use, but it has been the experience of the writer that these will only have to be increased from time to time.

Permanent results can be obtained by the advancement of one Externus. The refractive error, of course, should be corrected.

Case: Mr. L. S., age 38.

About five years ago, patient began to be troubled with attacks once or twice a week, very much resembling migraine. They would begin with slight vertigo, passing into intense headache and ending in nausea and vomiting. The headache was confined mainly to supraorbital and occipital regions. About 18 monhts ago, he saw an optician who prescribed Prisms base out, and for a time symptoms all disappeared. He is now afflicted with the same complaint in its original severity.

Examination shows him to be hypermetropic, taking under a Cycloplegic, a plus .50 D Sphere, with a small cylinder in each eye. There is an Esophoria for distance, of 12 Prisms Diopters, and for near, 5 Prisms Diopters. He has been wearing 2° prisms, base out, in each lens.

I did an advancement of one Externus only. Since that time, two years ago, patient has had no attacks and is very thankful. He complained of some difficulty in wearing his glasses, but this is gradually disappearing. Screen and Parallax test shows less than 1° of Esophoria for distance.

4. *Cases of Divergence Excess.*

This form of Exophoria, up to 8 or 10°, does not, as a rule, give rise to any asthenopic symptoms. The patient is aware of a transient crossed diplopia at times. For distance, all previously mentioned tests show a marked Exophoria which is much less for near. The Convergence near point is normal. Prism Convergence is normal. Prism Divergence, abnormally great, over 9°.

This condition is not benefited by non-surgical treatment. When the condition becomes marked, the ideal operation for its correction is a recession of the Externus, provided the patient's convergence is good.

5. *Cases of Convergence Excess With a Divergence Insufficiency.*

As a secondary change following Convergence Excess, the Externus quite often loses its tone, with a consequent loss in the power of Divergence,

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causing the condition commonly spoken of as Con-comitant: Convergent Squint. It is readily diagnosed by the Screen and Parallax test.

Three clinical varieties are drescribed:

(a) Occasional Squint.

(b) Constant Unilateral Squint, about 85%.

(c) Alternating Squint, about 15%.

In constant unilateral squint, there is generally a marked difference in the refractive errors of the two eyes, while in the alternating type, the refraction is much the same and the visual acuity practically normal.

The treatment of C. C. squint is something with which every ophthalmologist is familiar.

When a case is seen early, the refractive error, under atropin, should be estimated, and the full correction, whether hyperopic, myopic or astigmatic, prescribed.

In hypermetropia of large amount, it would probably be wise to diminish the finding under atropin by .5 D for constant wear.

The age at which children will take glasses lies somewhere between 20 and 24 months, so up to this time it will be necessary to preserve the vision in each eye. The mother is instructed to place a patch over the eyes alternately for two or three hours each day. If we see the case when one eye is constantly deviated, an effort must be made to restore, if possible, the sight in the deviating eye. To accomplish this, two methods seem equally popular. Occlusion, by means of a patch, of the fixing eye daily for one-third of the child's waking hours, or suspending the accommodation of the fixing eye by the instillation of one drop of a 1% solution of atropin daily.

Although we are told that atropin used continuously for a time has no permanent effect on the accommodation, personally I think I would adhere to the patch method if possible.

At the age of 20 to 24 months, the child will generally accept glasses, and the full correction with atropin is given (Sol. Atropin 1% in each eye, 3 times daily for 3 days). If at the end of 6 to 9 months, the glasses being constantly worn, there is no improvement, operative measures must be undertaken.

In these very early cases, where the condition is primarily one of Convergence Excess, i. e., where the deviation seems to be more marked for near, good results can be obtained by doing a Recession of both Externi. In the later cases, where Divergence Insufficiency has become an important factor, a double advancement of the Externi should be done. In all cases, up to 25 or 40° a double advancement seems quite sufficient. In cases over this amount, it may be necessary to do a Recession of the Internus of the commonly deviating eye as well.

6. *Cases of Divergence Excess and a Convergence Insufficiency.*

The divergent form of Concomitant Strabismus is comparatively rare. Dr. H. W. Wooton some time ago, in an article published in the Ophthalmoscope, drew attention to the relation which existed between the predominating condition and the error of refraction.

The cases of Divergence Excess with a mild degree of Convergence Insufficiency co-existing, he found to be mostly hypermetropic; and cases of Convergence Insufficiency with a mild degree of Divergence Excess, to be mostly myopic. Cases in which both conditions were marked, showed frequent anisometropia.

The determination of the muscular anomaly in any case is quite simple. In cases of Divergence Excess, the deviation exists only for distance and may disappear or become very much less for near. The power of Convergence can be shown to be unimpaired.

In Convergence Insufficiency cases, the squint is more marked for near. In marked cases of both, the squint will be present both for distance and near, and the power of Convergence, practically nil.

In the treatment of divergent strabismus, the refraction must first be

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corrected. Some of the myopic cases, where the myopia is fairly equal in both eyes and the Convergence Insufficiency is the predominating muscular anomaly, seem to respond somewhat to this treatment.

When Divergence Excess constitutes the predominating anomaly, correcting the refraction produces no effect.

It follows then that most cases must be operated upon to obtain a cure.

The Hypermetropic cases with Divergence Excess should be treated by doing free Tenotomies of both Externi. Tenotomy should constitute the primary treatment, even if a well-marked Convergence Insufficiency co-exists, and should be repeated until the eyes are straight for distance. After this procedure, the Interni sometimes regain a certain amount of tone, so it is wise to defer advancing one or both Interni until some later date.

The myopic cases associated with Convergence Insufficiency should be treated by advancing one or both Interni. In cases where the vision in one eye is greatly below par, advancement of the Internus and recession of the Externus of the deviating eye should be done at the same sitting.

This latter statement also applies to cases of divergence due to an over-correction obtained by the old method of tenotomizing the Internus for C. C. Squint.

Case: Mr. H. J. K., age 62.

At the age of 15 years, patient had a Tenotomy performed for the correction of a C. C. Squint. An over-correction followed, and in the course of five or six years the eye became widely divergent. On examination, there was a 40° divergent squint. Convergence is practically nil; his adduction, fairly good.

Vision: (right / (left with glasses:

The patient expressed a desire to have the eye straightened, so I did an advancement of the Internus with a recession of the Externus of the deviating eye. As the Internus was weak and atonic, I severed the Externus before attempting to advance it. After I had accomplished the advancement, I completed my recession of the Externus with a very satisfactory result. Screen and Parallax for distance shows no deviation whatever.

7. *Cases of Hyperphoria and Hypertropia.*

This condition is fairly common and is one of the causes of Frontal and Orbital Neuralgias.

All cases up to 5—8° should be corrected with prisms, the patient being given as much as he will take comfortably, divided between the two eyes. I find that they will accept one-half to two-thirds of the total error without any difficulty. Operation, in these cases, has not been satisfactory. If, however, an operation is to be tried, the one suggested is an Advancement of the Inferior Rectus of the higher eye.

8. *Cases of Paralysis.*

Under the heading "Paralysis" may be grouped any of the non-comitant forms of deviation, from a very mild Paresis to a complete loss of power.

The lesion may lie anywhere along the course of the nerve from its beginning in the cortex to its termination in the muscle itself, and may be caused by New-Growths, Vascular Conditions, Intoxications or Trauma.

The condition may be limited to one muscle only, or may involve a combination of muscles. Quite often, we see a paralysis affecting only certain branches of a nerve, while other terminal divisions escape.

In making a diagnosis of an affected muscle, we take into consideration:

(a) The Restriction of movement—determined by studying the action of the different muscles in the six cardinal directions.

(b) The deviation produced—determined by means of the Screen and Parallax test.

(c) The double vision, estimated by the use of the Tangent Screen.

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In the treatment of Paralysis, the results obtained by non-operative treatment depend almost entirely on the cause. Syphilitic and rheumatic cases seem to offer the best prognosis, and the results are exceedingly good if the usual treatment for these diseases be instituted early.

A fair percentage of the traumatic cases seem to recover during the first year following the injury, so it is always wise to observe any case for this period before deciding on an operation. During this term, electricity may be employed locally and a ground glass placed before the paralyzed eye, to relieve the troublesome diplopia and vertigo.

In old cases, where atrophy of the paralyzed muscle has taken place, a cure is practically impossible.

The operative treatment of the Paretic form is very satisfactory, especially where one muscle only is involved.

Case: Mr. J. W., age 38.

Patient is referred by a general man, complaining of Diplopia. Gives a history of being struck on the right side of the head with a piece of rock some three months ago.

Examination shows a large depression in the right Fronto-Parietal area. Examination of the eyes:

Vision 20/20 in both. There is a R. H. of 10 prism diopters and an Esotropia of 8. The right eye lags on looking down and out, and the patient states that the diplopia increases as we carry the vision into this field. A ground glass was prescribed for the right eye and the patient kept under observation for the balance of the year. At the end of this time no improvement had taken place and the patient was still unable to work on account of the troublesome diplopia. An advancement of the right inferior rectus was done, with a very gratifying result. The patient was greatly elated, being able to resume his regular occupation, quite free from diplopia. It was possible, however, to get a slight diplopia on carrying patient's eyes forcibly in the direction of the paralyzed muscle. But, for all practical purposes, he has not been bothered with diplopia and is quite happy.

Cases of complete paralysis do not behave quite so beautifully. The result of an operation, however, is an improvement. If the paralysis is complete, so that the paralyzed muscle is no longer able to exert any action whatever upon the eye-ball, it is only from a very extensive advancement that we can expect to get any effect, and even then the only effect we can gain is that the greatly deviated eye is brought back approximately to the Primary position. This position is not retained, however, as the strong healthy antagonist soon overcomes its atonic brother. It has been my experience that only a very complete tenotomy of the antagonist will allow the eye-ball to remain in the Primary position, free from motility in a lateral direction.

Case: Mr. J. A., age 45.

Patient's skull was fractured by being struck with a large piece of wood two months ago. Examination shows the sixth nerve on the left to be completely paralyzed and the eye to be markedly abducted. After a year had elapsed, I advanced the paralyzed muscle and did a Recession of the Internus. The eye was straight, however, only for a few days, and soon began to swing in on account of the powerful Internus pulling against its atonic antagonist. After a time, I again receded the Internus, but with only the same result. I then did a complete tenotomy of the Internus and the eye remained straight, with no apparent proptosis. He gets a slight amount of movement through the other muscles and avoids Diplopia by turning his head. I cite the case to show how impossible it is to gain results by attempting to advance a completely paralyzed muscle.

In cases of head tilting, more often seen in children, where there is a Paresis of the Superior Rectus of the one eye with spasm of the Interior Oblique of the other, the treatment suggested and carried out by White, of New York, is a complete Tenotomy of the Inferior Oblique. This almost certainly clears up the head tilting.

The *Advancement Operation,* which I have been using and which has

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been employed at the Manhattan Eye, Ear, Nose and Throat Hospital for some years, is the Wooton modification of the Worth operation. At the New York Eye and Ear Infirmary, however, they do nothing but the Reece Resection. Both are good, but personally I favor the former as it seems to me to be a true advancement.

The technique of this operation is, no doubt, quite familiar to you all. The conjunctival incision extends from 7 to 11 o'clock, about 1—2 m.m. behind the Limbus. The conjunctiva is elevated posteriorly over the attachment of the muscle, which is raised, with Tenon's capsule, on two hooks. Two lateral sutures are now placed, piercing the outer fourth of the tendon about 3—4 m.m. from its attachment, the needle being passed through twice in order to form a loop. The muscle is now divided behind its insertion. The central suture is begun by passing the needle through the outer layer of sclera, at about the center of the original conjunctival incision and parallel to it. The ends are now carried through the muscle from behind onward at a point 4 m.m. from the end revolving the middle third of the tendon. These are next carried through the conjunctiva at its edge.

The lower of the two ends of each of the lateral sutures is now passed through the outer layer of the sclera at 11 and 7 o'clock respectively. The stump of the tendon, which up to this time has served as a holding point for the fixation forceps, while the scleral sutures were being placed, is now removed. The eye is forcibly turned in the direction of the muscle and the sutures tied. Following the operation, both eyes are kept bandaged from 10 to 12 days. The eyes are dressed separately once daily, Argyrol and Atropin being instilled. At the end of this time, the patient is given his correction which must be worn on the removal of the bandage. The stitches generally fall out themselves. If they still remain, they may be removed at this time.

The *Recession Operation* was devised about five years ago by Dr. P. C. Jamieson of Brooklyn, and published in the "Archives of Ophthalmology" (Vol. 4, L 51, No. 5, 1922). This operation, with certainty, fixes the attachment at any point desired.

Since this time, modifications have been made, but the idea still holds good. The conjunctival incision is made across the insertion of the muscle, following the semilunar fold, about 7 m.m. in length. The muscle is exposed and raised with Tenon's capsule on hooks, as before. One double armed suture only is used. Both needles are inserted into the center of the muscle, about 2 m.m. behind its insertion, and carried in the substance of the muscle, one emerging at the upper edge and the other at the lower edge of the tendon. An extra turn is taken around the outer fibres with each suture, in order to keep the muscle spread. It is now severed about 1 m.m. behind its insertion. The needles are now dipped through the outer scleral fibres, at the new point of attachment, the width of the tendon apart. From this point, they are carried through the old stump of the tendon and then through the anterior edge of the conjunctiva and gently tied. The conjunctival wound is closed by interrupted sutures.

GENERAL CONSIDERATIONS

1. The success of the operation performed will depend to a great extent upon a proper diagnosis of Insufficiency or Excess, or a combination of both.
2. An advancement of any of the ocular muscles does not cause diplopia and is consequently much safer than a Tenotomy.
3. Where vision is good in both eyes, a Tenotomy should not be done except in the form of a Recession.
4. In a case of Paresis or Under-action, the weak muscle should always be advanced.
5. In a case where there is a vertical as well as a lateral deviation, the correction of the former often alleviates to a great extent the lateral deviation.

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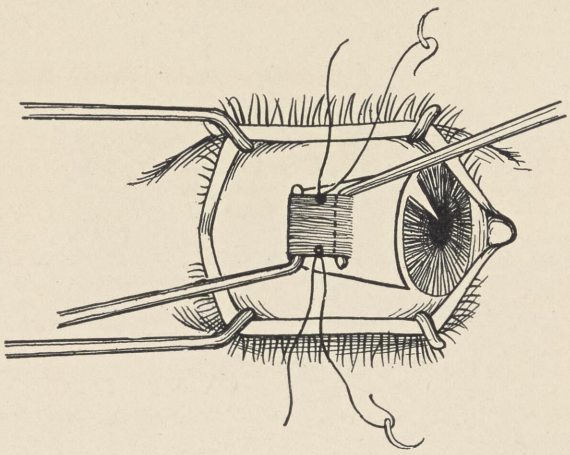
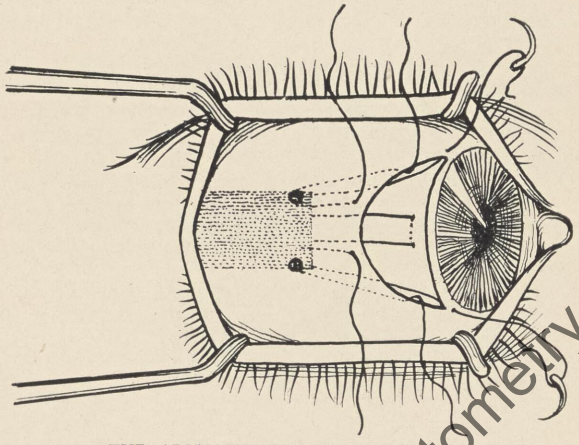


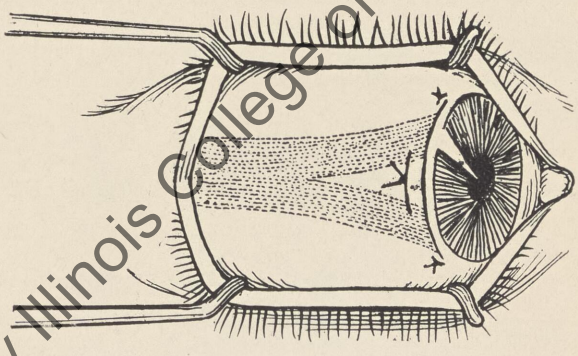
THE ADVANCEMENT OPERATION (1)

The muscle is shown raised on hooks and the side sutures placed. These are passed through in the form of a loop, and embrace the outer fourth of the Tendon.



THE ADVANCEMENT OPERATION (2)

Shows the three sutures placed. The central suture is passed through the outer layer of sclera first and the ends brought back through muscle and conjunctiva.



THE ADVANCEMENT OPERATION (3)

The sutures are tied, giving the muscle a fan-shaped attachment.

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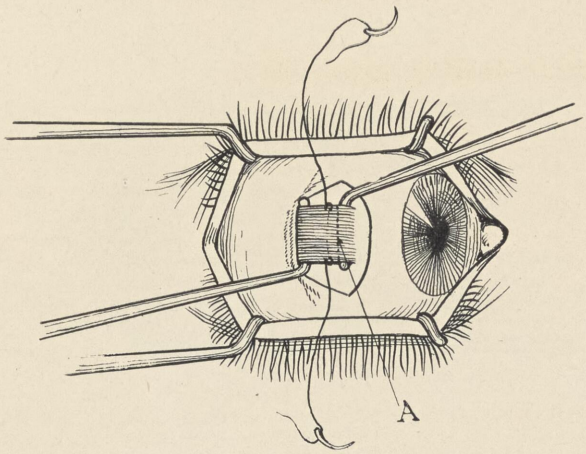
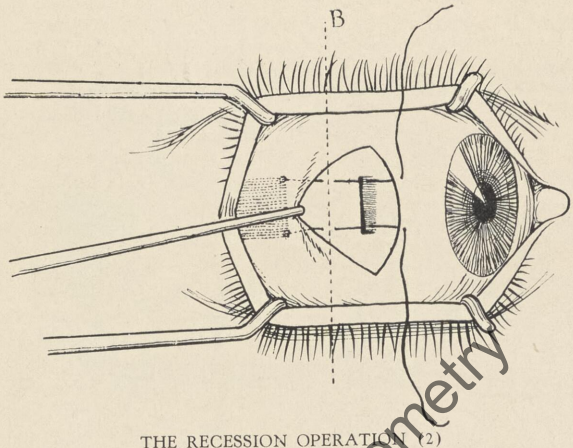


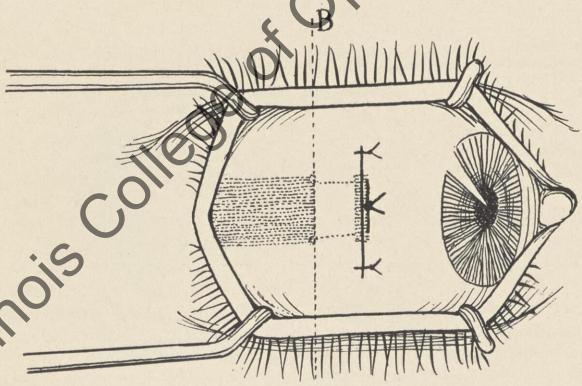
THE RECESSION OPERATION (1)

The single double-armed suture is begun in the center of the muscle, and carried in its substance to each border where an extra loop is made to prevent the muscle from bunching. The muscle is severed at A.



THE RECESSION OPERATION (2)

B represents the site of the new attachment, so the needles are dipped through the outer scleral fibers at this point, then through the old stump of the muscle and the anterior edge of conjunctiva.



THE RECESSION OPERATION (3)

The end result. The wound is closed by two interrupted conjunctival sutures.

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6. In the correction of a convergent squint of high degree, a Recession should not be performed at the same time as the Double Advancement, but at some later date, when the required amount can be properly estimated.
7. Duane says that in Heterophoria, an operation—if performed with judgment and with a careful regard to the underlying conditions of the Heterophoria—often relieves, sometimes to a surprising degree, the asthenopic confusion, headache and other symptoms. In squint, the results obtained are only cosmetic, and in only a few instances is binocular vision restored.

Discussion opened by Dr. J. Thomas Dowling, Seattle:

Ocular deviation is a subject of great interest to me and, I presume, to all of the eye men. It is a subject that we are interested in scientifically and one that every eye man has a great deal to do with. It is also a subject that we have to take into consideration in making our living which, to most of us, is quite an important consideration.

The first thing I thought of in connection with Dr. Smith's paper was the necessity of very careful diagnosis as to the type of squint. Before we can do anything in the treatment of squint we have to know pretty definitely what type of squint we are dealing with. Squint, as you know, varies from the one anyone can see to one that takes a very careful, thorough and painstaking examination. Another point which is very important in the diagnosis, and in the treatment, is a careful measure of the refractive error. This must be very carefully done under a mydriatic and sometimes may have to be done two or three times as a check. I often have it checked by some other man in whom I have confidence and I feel that it is very important that this checking be done. As to the age of the patient, the treatment cannot begin too soon, even as early as the third year. This necessitates great cooperation on the part of the patient and a very great cooperation on the part of the mother or attending nurse or whoever takes care of the little individual. This cooperation is often very difficult to obtain and a good deal depends upon the attitude of the doctor, but with all you can do in the meantime, the parents often tire of your so-called non-operative treatment.

Doctor Smith has divided treatment into two classes, operative and non-operative. Non-operative treatment Doctor Smith has outlined in a very comprehensive manner and has gone over it very thoroughly from a scientific as well as from a practical standpoint.

We all try glasses for the correction of refractive errors, the use of prisms for exercise and, in all cases, Stereoscopic instruments are used in an endeavor to improve the condition. All of this is tried after a certain length of time, depending entirely upon the judgment of the physician as to whether the vision is holding its own. By that I mean that the refractive error is not changing very much in either eye. If there is a marked change, we are losing ground and it is then time to go ahead with the operative treatment. I am frank to admit that I have not had as much experience as I would like to have had but I find in following up the cases of other men in my locality, as well as in the large Eastern centers, that the operative treatment sometimes is not quite all that the individual operated upon anticipates and not quite all that the doctor expects. This happens even in the hands of the best operators so we need not feel badly if sometimes we do not get the most satisfactory results. Another point in operating on squint, the judgment of the operator must, of course, come in very strong and one should not try to do too much operating at one sitting as there is plenty of time to operate on the individual the second time and even, if necessary, the third time. I believe that this is the accepted viewpoint of the more experienced men of New York, Boston and large Eastern centers.

Doctor Smith has touched upon injuries in his paper. It has been my experience in injuries to not operate too quickly after the injury because the patient often recovers without operation. It must be remembered that a

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squint in an injured man may be the result of lues or some other acute or chronic disease. All laboratory work should be done very carefully and the treatment must be the result of judgment. Do not operate too quickly after an injury, is my opinion, and I am glad to know I agree with Doctor Smith.

I want to say I enjoyed Doctor Smith's paper very much. It is scientific and, I might say, practical, which nowadays is very important as we hear many scientific papers and after hearing them wonder what it was all about. I am not decrying the fact that papers should not be scientific, but I think in a meeting of this kind where we are here to learn from each other, the papers should be practical as well as scientific and such a paper is Doctor Smith's.

Dr. C. A. Veasey, Spokane, Wash.:

A probable reason for not finding more cases of orthophoria is that patients presenting themselves for eye examinations and complaining of ocular discomfort are found to possess some refractive error. The disturbance of the relation between accommodation and convergence will account for at least a part of these.

It is our custom to pay very little attention to the lateral deviations of the ocular muscles unless they are producing symptoms. Our first effort is to correct the refractive errors and to place the patients in the best possible hygienic conditions, paying especial attention to the correction of faulty habits. If the ocular symptoms persist, orthoptic exercises are employed for deficient converging power, if present. Many patients have lived indoor lives and are more or less neurotic, and in such we find that a vacation in the open oftentimes assists materially in the relief of the ocular symptoms due to the orthophoria. It must be remembered that one can possess a considerable degree of esophoria or exophoria if in good health without ocular symptoms or ocular discomfort. As a last resort before operative procedure, prisms may be employed.

It is always our custom to correct fully all amounts of hyperphoria after repeated examinations have determined definitely the amount existing. For many years we have employed the cover test as an aid in determining the amount of latent heterophoria.

Dr. Otto Barkan, San Francisco:

I was much interested in Dr. Stookey's definition of physiological muscle balance. In addition to what has already been said, I should like to stress the importance of the individual's nerve reserve. It is a common experience that Europeans with muscle errors which had been latent abroad develop symptoms shortly after coming to the United States and being exposed to the high tension of our metropolitan life. The same obtains in our own people who come from the country to the large cities. The errors are decompensated when the individual's supply of nerve energy becomes depleted and he is forced to call upon his physiological nerve reserve. Acclimatization and proper advice will often result in compensation of these errors and thus relieve the patient of his symptoms.

Dr. J. G. Parsons, Lewistown, Montana:

I believe there has been no mention made, so far in the discussion, of the bearing of sinus involvement upon muscular unbalance. There is abundant evidence based upon good anatomical reasoning, to show that ethmoidal involvement may cause a convergence insufficiency. By reason of the close relation of the internal recti to the orbital plate of the ethmoid, intranasal inflammations in this region may may make themselves felt in a "soreness" or "lameness," if you please, of the muscles. Ocular discomfort in acute and chronic ethmoiditis is largely explained in this manner. Application of adrenalin gives a temporary relief—pointing toward the probability of the relation.

I believe we should not overlook the pathologic ethmoid in considering our treatment of muscular unbalance.

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Dr. Hamilton Stillson, Seattle, Wash.:

When Einstein suggested that the Pythagorian Theorum was not exactly true we all wondered what Einstein was driving at. "Why, all of our calculations in Physics are based on the 'pons asinorum'." Similarly, when one suggests that Donders' "Orthophoria" is a mere term convenient for academic discussion, he is considered quite heretical. Yet surely we have all realized that parallelism of the visual axes was, for infinity, from anatomical considerations, not only highly improbable but was never found in fact. Who has ever seen a case of orthophoria?

Orthophoria is a convenient myth we have all accepted with complacent tolerance.

Dr. A. W. Howe, Tacoma, Wash.:

As I remember, Doctor Smith made a statement that one should not overlook the ocular deviation that might be caused from intra-nasal conditions.

Doctor Smith being from Lewistown, Montana, recalls to me a case that I saw who came from Lewistown, Montana, in February, 1921, while I was practicing in Minneapolis. The patient gave a history that a month or so before coming to Minneapolis his right eye began to turn outward, the upper lid began to droop, and occasionally he saw double. Every morning upon arising a certain amount of mucous dropped back into his throat and he was forced to clear it. This latter condition, he said, had existed for many years but had been worse the last two or three months. He also gave a history of having his nose fractured when a child three years of age and could never breathe through the right side of the nose. A thorough physical examination including blood Wassermans were negative. The sinuses were negative as far as one could make out from the X-rays. The antrums were negative on irrigation. A submucous resection was performed. In forty-eight hours after the operation the double vision disappeared, his eye became straight, and the dropping of the lid disappeared. Two years after this, the case was followed up and the man had no recurrence of the trouble. Our diagnosis was a mucocele of the right ethmoid and pressure due to high septial deviation.

Dr. A. J. Smith, Vancouver (Closing):

In closing, I would like to say that I quite agree with Dr. McCool's suggestion as to the Accommodative and Non-accommodative types of Convergence Insufficiency, and the proper general treatment for the latter condition. This is an important factor and one which should not have been omitted.

Dr. McCool also states that he has not obtained satisfactory results with the head tilting in cases of Paresis of a Superior Rectus with spasm of the Inferior Oblique by Tenotomizing the Inferior Oblique, but he has obtained results from an advancement of the Paretic Muscle.

I must say that this has not been my experience. In several cases operated upon by Dr. White of New York, the head tilting cleared up almost immediately.

Advancement of a Superior Rectus, on the other hand, does not seem to be a satisfactory operation, for many of these operated cases afterwards complain of asthenopia on looking down.

As to the safety of the scleral sutures, I have yet to hear of any intra-ocular infections resulting from them.

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VISION IN RELATION TO INDUSTRY*

E. M. NEHER, M. D., Salt Lake City, Utah

The condition of the eyesight of employees is the most important factor affecting the output of the industries of our country. Until very recently it has received little consideration, excepting tests for the acuity of vision and color blindness by the railroads and a few of the larger manufacturers. By recognizing their duty in determining and conserving the vision of the employees, the corporation not only becomes contributors to the health and upbuilding of society, but they find production is increased, the quality of workmanship is improved, less material is wasted, fewer accidents occur, greater individual effort is made possible and above all, there is a better morale, greater comfort and happiness among the employees when off duty, as well as when at work.

Physicians as well as an increasing number of employers are unanimous in the opinion that the eyesight of the workman should be conserved, but it is difficult to find a satisfactory method of accomplishing this. In my opinion the methods should include:

1. Legislation.
2. Vision testing and proper correction of defects.
3. Protection of eyes against injuries.
4. Education.

Intelligent legislation—that which is fair to both employee and employer —is the primary factor in inaugurating safety measures. This is accomplished best by establishing an Industrial Commission which is given full power to require the necessary accident reports, to make and enforce safety measures and to administer equitable compensation, rather than to wait on the slow wheels of a political legislature which meets once in two years.

Proper eye examinations should be made of new employees and frequent re-examinations of old ones. Systematic testing of vision has not been generally done, as is shown by the most accurate statistics I was able to obtain. The Eyesight Conservation Council of America sent inquiries to a selected list of seven hundred and fifty companies and commercial establishments, having a safety and a welfare department. Replies were received from one-third the number but some were too meager to be useful and only one hundred and seventy reports could be analyzed. Of this number ninety-five companies stated eye tests were given, but only twenty-one companies reported periodic examinations. Forty companies, representing twenty different kinds of industries, located in various parts of the United States, gave the result of eye examinations of 204,817 employees. The tests used ranged from the most superficial to the more thorough, with an average of 44.3% with defective vision. The Buick Motor Car Company found that 56% of applicants for employment had subnormal vision while 48.5% of the sixty thousand Ford employees examined had lowered visual acuity. Seven companies who made a more thorough examination of the eyes of 12,800 employees, found 72% with errors of refraction. This study and report of examination of nearly 300,000 employees is the largest group that has ever been studied and the results are probably a fairly accurate incidence of the defective vision among the 42,000,000 persons gainfully employed in the United States.

The survey showed that in the group of 204,817 examinations only 19.6% were wearing glasses and many of these corrections were wrong. One company found two-thirds of the glasses the employees were wearing to be incorrect. In the report of seventeen companies, employing 83,825 persons, less than 10% were wearing glasses while 49% had defective vision. A concrete example of corrected vision improving production is shown by Whiting and Davis Company of Plainfield, Mass., who increased the output of their factory 27% after they had carefully corrected errors of refraction among the employees.

* Dr. Neher having been delayed, Dr. Hampton read paper.

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The Public Health Service made a series of visual acuity tests at different ages and found the percentage of persons with normal vision increased from six to eighteen years of age and then gradually decreased to forty-five, after which there was a rapid decline due to lowered accommodation, sclerosis of the crystalline lens and pathology in the fundus incident to advancing years. Increasing age especially lowers the vision for close work and in all examinations, both the distant and close vision should be recorded.

While we have no statistics showing the definite relation between defective vision and accidents, we may be sure that for our efficiency and safety, good vision both central and peripheral is the most important of our special senses.

The presence of defective vision, its causes and remedies, can be determined only by competent physicians. Each worker is a unit of the great industrial machine and his mental and physical fitness, including proper vision, should be determined by examination for the position for which he has applied, in order to secure his own safety as well as the safety of others. Some employees and labor organizations are adverse to physical examinations. Nothing is more absurd. Such objectors are enemies of right and justice and the safety of fellow workmen.

The visual requirements, both with and without glasses, necessary for the different classes of positions are not here being discussed. It is a great injustice to an applicant for the employer not to take into consideration his vision as corrected by glasses. However, since many State Industrial Commissions do not reckon the acuity of corrected vision when naming the amount of compensation, the employer may not want to employ a man with subnormal vision. A rule is said to be a poor one which will not work both ways. Since it is shown by statistics quoted in this paper that from 44% to 72%, depending upon the thoroughness of the test, have defective vision without the use of glasses, is it right and just to employer or insurance carrier to assume every injured eye had normal vision previous to the accident and to make the compensation award on this basis? As example, in my own practice, a man forty-eight years of age had a small foreign body embedded in his cornea. At the time of removal it was noted that his vision was very low in the injured eye. Examination of the fundus showed an old rupture of the choroid. The man denied any previous injury until the X-ray showed twenty-five small metallic shadows embedded in the skin of the face. This, he then admitted, resulted from an explosion years previous to his present employment, at which time the choroid was undoubtedly injured. Should the man be paid for the loss of his eye on account of the recent foreign body in the cornea? It is just as reasonable for a man who receives a scratch on the stump of a leg which was shot off during the World War, to be compensated for the loss of his limb on account of the recent injury to the stump.

EYE PROTECTION

The protection of the eye against hazards should be a most important factor of the safety department of every industry. A great many of the eye protection devices must be worn by the workmen in the form of glasses or goggles. The type of eye protection device to be used depends on the nature of the work to be performed. The National Safety Code for the Protection of the Head and Eyes of Industrial Workers, published in No. 2 of the Bureau of Standards Hand Book Series, divides the different operations or processes in which it is necessary that the operator be given protection for the head and eyes, into nine groups, each of which requires a protector having distinctive features. These types were selected as the result of the experience of a number of physicians, safety engineers and goggle manufacturers. There has been a natural adverseness by the workmen to the use of goggles on account of alleged inconvenience and discomfort. These supposedly objectionable features can be largely overcome by having them fit properly and their refractive errors ground in the glass. Naturally if the workman has a defect of vision, unrecognized but handicapping him to some degree, the plain glass

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TABLE I

UTAH STATE INDUSTRIAL COMMISSION

REPORT OF NINE-YEAR PERIOD ENDING JUNE 30, 1926

			Compensation	*Medical*	*Total*
Total Number of Accident Cases	101,463		$5,547,107.00	$1,514,638.00	$7,061,745.00
Total Permanent Disability	19	Estimated Cost $15,000 each			285,000.00
Total Permanent Disability Eye Cases	16	Estimated Cost 15,000 each			245,000.00
Total Permanent Partial Disability	1,359		1,019,317.00	665,555.00	1,684,872.00
Total Permanent Partial Disability Eye Cases	72				
Partial Loss of Vision	62		36,681.37	8,558.00	
Entire Loss of Vision	87		141,214.45	19,496.09	
Loss of Eye (enucleation)	73		128,096.60	14,863.73	348,913.24
Temporary Eye Injuries	7,321	(Estimated)	7,321.00	21,963.00	28,284.00
Total number of Eye Cases	7,560	With a Total Cost of			622,197.24

would apparently serve to emphasize this defect. Correct refraction in the glass solves this objection. Properly corrected vision lessens headaches and nervous irritability, lessens loss of time and energy and obviates eventual loss of sight.

The absence of proper eye protection in industries is shown by the great number of eye accidents reported. The National Safety Council estimates 250,000 eye injuries annually. In California, during the four-year period of 1917 to 1921, of the 9328 permanent injuries reported, 13% involved the eyes. The American Foundry Association reports that 20% of all nonfatal accidents occurring in foundries, are eye injuries, of which 28% resulted in the loss of one eye and 10% in the loss of both. This same report states that 95% of the accidents could be prevented by the use of goggles and other protective devices.

There are far too many eye accidents in the industries of Utah. In both the metal and coal mines, we find the men especially adverse to wearing goggles, stating the perspiration and air currents cause the lenses to fog. These and other objections can be overcome by using the correct type, by wearing a sweat absorbing band around the forehead, and coating the lenses with one of the various substances used to prevent the glass from fogging. The use of the proper eye protective devices by the employees of Utah would reduce the eye injuries ninety per cent. That this would be no small item is shown by statistics I was able to obtain from the State Industrial Commission, through the kindness of the secretary.

See Table I.

During the nine-year period of the existence of the Industrial Commission of Utah, ending June 30, 1926, there was a total of 101,463 accidents, with a paid compensation of over five and one-half million dollars and medical fees exceeding one and one-half million, making a total cost of over seven million dollars for the accidents. In this number are included nineteen totally permanently disabled, of whom sixteen (84%) have eye injuries with an estimated cost of $15,000 each, or a total of $245,000. There are thirteen hundred fifty-nine persons partially permanently disabled, two hundred and twenty-two (16.3%) of them eye injuries, with a cost in compensation and doctor fees of nearly $350,000 (21%) of the total cost of this class of injuries. In addition there are seven thousand three hundred and twenty-one temporary eye disabilities, at a cost of $28,000. This makes a total number of eye injuries seven thousand five hundred and sixty (7.5%) and costing nearly $625,000, or 8.7% of the total amount expended by the Commission. Yet this huge sum is insignificant when compared to the great amount of suffering, serious disability and great loss, not only to the unfortunate employee but also to those depending upon him for support and comfort. How appalling these facts are when we realize that nine out of every ten of these accidents could have been prevented by the use of the proper eye protecting devices.

EDUCATION

Great possibilities for elimination of the destruction of vision lies in education. Educate the State to realize its humanitarian and economic obligation, by establishing and enforcing adequate safety-first measures. Safety inspectors should be of sufficient number to supervise small plants as well as the large. They should be not only capable of knowing their duty but should have the backbone necessary to enforce safety measures.

The management of industries should be instructed in the economic value of eyesight conservation. Every official should be on the alert and willing to provide necessary safety appliances to protect the eyes of their employees. They should appreciate that every dollar intelligently invested in safety devices means a saving of many dollars in compensation and a continuation of uninterrupted service.

Education of the employees, especially of the so-called "hard boiled" type

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and the ignorant laborer, is difficult, but if all the companies will require the observance of safety measures, he will soon fall in line. The following suggestions may be helpful: Bulletin boards judiciously used. They should be placed where men frequently see them, and it may be necessary to have the bulletins printed in other than the English language. The most effective bulletins are those made from experiences within the plant; for example, a photograph of a fellow workman, together with a pair of safety goggles which prevented injury to his eye. Beside it place the picture, with appropriate description, of an unfortunate man who lost an eye because he did not wear the proper protection. Bulletin boards can be used to place warnings, as a warning against the danger of infection from allowing a fellow workman to remove a foreign body from his eye with a match or a soiled handkerchief; warning against danger to the eyes of an observer who may be watching an electric welder or cutter at work.

Safety bulletins may be posted, such as:

"The man who loses one eye, sometimes wears the other out looking for a job."

"Once we knew a man who could not see the need of wearing goggles and now he cannot see the goggles."

"Protect your eyes and save yourself a life of Darkness."

"Two good eyes are all you have. Save them."

In conclusion, the science of human rehabitulation has developed artificial hands, arms and legs which can do almost anything the human members can do, but no one has developed an eye that can see, nor do we even dare to predict the day will come when it will be possible to replace the human eye with one that is capable of interpreting rays of light. This fact alone makes the protection and conservation of vision in industry a most important subject.

Discussion opened by Dr. A. W. Morse, Butte, Montana:

It is rather a difficult thing to discuss so exhaustive a paper as this. It seems to me the thing that Dr. Neher wishes to bring before us is the fact that each one can do his part in helping to accomplish this conservation of vision. We are the ones who see these men and have to do with the companies who arrange for their examinations. With the corporations and insurance companies, the economic consideration will bring about more thorough examinations. As brought out in the paper, when they have to pay for the loss of vision which may have existed before the injury, but which cannot be proven, the companies will insist on having at least a record of vision when the applicant goes to work. The increased production and fewer accidents, when proper lenses are worn, will several times pay for these corrective measures. If all the companies should immediately request this service, I do not believe that the medical profession would be in a position to furnish it. The fact that so large a proportion are incorrectly fitted with lenses emphasizes the fact that the work needs careful attention by competent men. I am quite sure that in the long run it would be cheaper and more satisfactory to the companies, as well as the men, to have the work done by competent men, and adequately paid for. We owe it to ourselves, as well as to the men directly involved, to understand their problems, and have constructive suggestions to offer whenever this subject permits discussion. We must prepare to undertake this work when the need is understood.

We commend Dr. Neher on his vision and leadership in this important trend of the times.

Dr. P. C. Means, Santa Barbara, Calif.:

As perhaps some of you know, the Department of Commerce is preparing to carry out regulation of commercial flying. Planes, landing fields and flyers will be periodically examined. All flyers will be given a very stiff physical examination with special care to the eyes and ears, before a license is issued. Re-examination yearly. Every locality where there is any flying

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activity will have an examiner. To those interested it should prove a desirable appointment and is especially so to eye, ear, nose and throat men.

Dr. E. M. Neher (Closing):

To see that eyes are thoroughly tested and regularly tested and accurately tested. As proven by statistics nine out of ten can be prevented and production can be increased 25%.

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VITAMINS AND DIETETICS IN RELATION TO OTO-LARYNGOLOGY

BURT R. SHURLY, M.D., Detroit, Michigan

What can this great medical profession and its highly specialized experts do to reach far underneath the problems of surgery, medicine, and oto-laryngology, and analyze our problems of infection and immunity, prevent the ravages of suppuration, of physical deformities and deficiencies, and increase the span of life.

A world of work has been done and many startling scientific facts have been brought to the light. If one-fifth of our profession are intimately interested in disease of the eye, ear, nose and throat, if more than 70% of all focal infection is in the tonsils, sinuses, and teeth, and if the great percentage of 22,000,000 school children fall within the domain of our special work, is it not fitting and necessary that our knowledge and interest should be devoted extensively to a solution of the etiologic factors and the prevention of these alarming conditions.

It is therefore of eminent importance that oto-laryngology throw its influence and scientific ardor into the realm of related bio-chemistry and the research problems so closely interwoven with our scientific progress.

The bio-chemic relation of lowered resistance to infection and temporary and increased loss of hearing in the presence of fatigue was demonstrated in my day at the University of Wisconsin under the direction of Professor C. F. Hodge. Many carrier pigeons were examined by cross section of the spinal cord after periods of long flight and after periods of resting and full nutrition. The proto-plasmic granules of the giant cells were completely absent after flight and completely filled the cell after good nutrition and rest. It would appear as if the sal-ammoniac in the battery was diminished and the spark was low.

While laboratory work has done perhaps more than any other branch of science toward clearing up many vexed questions about physiologic and pathologic activities, yet to be of lasting value and guidance it must agree with general and clinical observations. While this has been the case more often than otherwise, there are instances where laboratory observation and clinical data have crossed swords.

It is quite possible to realize the intimate relationship of infection in Waldeyers ring and the lymphatic system, to remote and general systemic infection. When we perform the experiments of Cushing who demonstrated by lumbar puncture and ventricular puncture at the same time that an indicator injected into the rectum could be detected in the cerebro-spinal fluid in 45 seconds and finally in the ventricular dripping 1½ minutes later.

The realm of Dietetics in oto-laryngology can be given no greater emphasis than in guarding the tonsil and adenoid child with marked lymphatism in its development, especially after operation. The experiments of Cramer, Drew and Mottram, showing the relation of Vitamen A and B to lymphoid tissue are interesting to us. They found that absence of Vitamen B from the diet of rats led to atrophy of lymphoid tissue throughout the body and also to lympho-penia. Polymorphonuclears were not affected. The absence of Vitamen A did not lend to atrophy of lymphoid tissue and there was no leucopenia. Absence of B led to loss of weight, emaciation, abnormal temperature and what is termed marasmus. The condition can be produced by X-ray exposure and a destruction of the lymphocytes that play an important role in the nutrition.

My interest in the nutritional problems related to our specialty was greatly stimulated by a case of scurvy that entered the Detroit Eye, Ear, Nose and Throat Hospital. The case exhibited the factors of faulty diet, weakness, mental depression, hemorrhage from the gums, ears and nose, with a purpura and subperiosteal hemorrhage and tenderness. The diagnosis was confirmed

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by the series of blood counts and the rapid recovery with iron and calcium intravenously and by mouth, together with abundant diet of meat, fresh fruits and vegetables. The antiscorbutic vitamen water soluble C., found particularly in oranges, lemons, limes and tomatoes, with Vitamen D., in red bone marrow, with a tonic rich in calcium and containing malt, pulverized ossis, lactic acid, pancreatin and pepsin, was continued for some months. At the present time, two years later, the patient is well.

Raw milk is the most satisfactory single article of food taken by man. It supplies the deficiencies of grains, tubers and fleshy roots. It is rich in calcium and phosphorous. No other foods supply these essentials in quantity, except leafy vegetables. Serious damage to bone structure in the nose, face and elsewhere, take place unless the diet is properly constituted.

The experiments of Kaufman, Crickmur, and Schultz assert that in Ricketts in rats there occur abnormalities of the osseous capsule of the internal ear which are identical with those changes in the long bones characterized in Ricketts and may interfere with the auditory function. Stepp and Fridenwald produced both Ricketts and Xerophthalmia by a diet very low in Vitamin A and phosphorous, but rich in calcium. Non-Richitic infants and young children have between 10 and 11 mg. of calcium and about five mgm. of inorganic phosphorous per 100 gms. of serum. All children under 2½ years with less than 3 mgm., of phosphorous had active Ricketts. Kramer, Casparis and Howland report the increase of phosphorous with the quartz mercury light is the equivalent of cod-liver oil. Orr, Hotl, Willin and Borne believe that cod-liver oil and the quartz light promote absorption of calcium and phosphorous from the intestine. Neither therapeutic aid however meets the defects in the composition of the diet directly or indirectly by supplying calcium or phosphorous, but by raising the potential of cellular activity and cell selection to influence ossification and calcification. Goldblatt and Scrames observe with others that liver taken from irradiated rats is growth producing, whereas the livers from non-irradiated rats is inactive. The same was found true of lung and muscle tissue. It is demonstrated that experimental Ricketts may be produced in rats by an inadequate supply of Vitamen D and disturbance of the calcium and phosphorous relation increasing with indigestion.

The daily problems of the oto-laryngologist bring him intimately into the realm of the dentist and some of the pertinent questions of caries and pyorrhea which seldom occur together. Why do some persons escape? Is there a definite relation between diet and decay. For many years it was believed that caries was purely of bacterial origin. The propaganda of mouth hygiene has ignored some essential facts.

Faulty diet and tuberculosis have something in common. It is very prevalent in Laborador where out door life prevails with continuous ventilation, but where a poor diet is the rule. It is essential that the protective foods, milk, leafy vegetables and fresh fruits, shall be taken in good quantity. This is important for the reason that the average American diet is deficient in calcium, over-rich in phosphorus, often lacking in Vitamen A and in some C, as well as certain fat found with D.

One of the most interesting clinical studies in Dietetics was made by McCollum in an institution of 234 with 84 children between the ages of 4-10 who were suffering from malnutrition. They were divided into groups and a quart of milk a day added to the regular diet. The results were phenomenal when added to the faulty and ricketty diet selected from cereals, tubers, roots and meat.

Sound teeth according to palentology, existed before the toothbrush, but the great underlying principle remains within the domain of diet. In the expectant and nursing mother and the infant, calcification begins between the fourth and fifth months of pregnancy and the first permanent teeth in the last third of prenatal life. We watch over the ears, nose and throat and mouth of infants and children and how keen is our interest to study with the

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pediatrist or without him if not available, the dietary problems of the individual case.

Rypins, of Kansas City, examined 1197 children between 3 and 6 years and found 27.2% had dental caries. Millanby, 1923, demonstrated that a substance like Vitamen A has a potent influence in the calcification of the teeth. Puppies, fed during the period of development of the permanent teeth, on deficient Vitamen A, showed one or more of the following defects: 1. Thick and poorly calcified jaw bones and alveolar processes. 2. Irregularity in the arrangement of the teeth, especially the lower incisors. 3. Delayed eruptions of permanent teeth. 4. Interference with the calcification of the enamel, often deficient in amount or badly formed. 5. Interference with the calcification of the denture. 6. Abnormal development of tissue at the gingival margins and the peridontial membrane. They continued these experiments in children with the questionable deduction that treatment has a very injurious effect on teeth.

The development of sound teeth with resistance is essentially a dietary problem. The small jaws and crowded teeth with tonsils and adenoids belong with faulty skeletal development and are influenced by dietary errors that favor Ricketts. Malposition also favors food packs and foci of fermentation.

Among the Italian children of the New York tenement district between 1-8 years, one would seldom see a child who was not bowlegged. The teeth of Italian immigrant children arriving in Boston were far superior to those of like age raised in the United States.

Research in the field of nutrition applied to preventative oto-laryngology offers an opportunity to raise the vitality of all mankind. The errors of diet may not cause deficiency disease, but insidiously their gradual operation more or less constant may be responsible for undermining the resistance in a degree so masked that the menace is quite unappreciated. As a cause of physical inferiority, instability of the central and vaso-motor nervous system, lack of recuperative power and continued fatigue, loss of endrocrine balance and lowered resistance it stands out alone in the first rank.

I have used hypodermic iron citrate for many years in lowered resistance, poor appetite, adenitis, and secondary anemia associated with focal infection. The haemoglobin will rise 5% a week. This average was maintained in a large series of cases where daily doses were given for twenty days. The Italian preparation containing .01 citrate of iron, soda glycerophosphate, sod. arseniate and strychnine is of great value in selected cases. The tonic action on the vaso-motor nerve centers gives the circulation to the glands and mucous membranes renewed vigor. In children, after tonsillectomy or in preparation for the same, I introduce iron, phosphorous and calcium, by the use of a sandwich three times a day with meals, made by spreading bread and butter with saccarated iron and pulverized bone in quantity sufficient to cover a fifty cent piece. The child prepares this sandwich at meal time and the importance of growing with properly selected food is given the valuable psychological impression this important subject deserves.

In feeding rats, Evans and Bishop found that green leaves produce a profound affect on cell reproduction, because of Vitamen E. The recent findings of McCollum that the function of Vitamen E, in some curious manner, is associated with iron assimilation confirms these clinical observations that I have made for years. A diet of liver with hypodermics of the green citrate or iron furnishes a combination rich in Vitamen E, with increased value in assimilation and cell reproduction.

Eggs are deficient in calcium, high in Vitamen A. It is estimated that six or seven eggs are equivalent to a quart of whole milk in Vitamen B. Sea foods, such as oysters, add to the iodine and Vitamen content.

In China and other parts of the Orient, where green leafy vegetables constitute a very important part of the food supply, Ricketts is almost unknown. Whereas, in Europe and America, where the tendency is to derive a large part of the food from milled cereals, meats, legume seeds and tubers, Ricketts and its frequent deformities, in the nose and mouth are common.

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In 1747, James Lind, a British naval surgeon, discovered that scurvy could be cured by the use of orange or lemon juice with the food. An interesting account by Dupain, 1920, details what happened in a large boy's school in England, during the winter term. Work and play fell far below normal. The boys became listless and irritable and many minor complaints developed. Although they were well fed, nothing was supplied in uncooked foods with few greens. Fresh fruit was added to the dietary and the trouble ceased. Incipient scurvy from a lack of Vitamen C was remedied.

While man requires Vitamen C, rabbits, pigeons, ducks, geese, turkeys and guinea fowl do not require it. Contact of food with copper utensils will destroy Vitamen C, as reported by Hess in experiments with milk. Delf says that the juice of a medium sized orange or lemon or a fairly large tomato, daily, furnishes sufficient Vitamen C for an adult.

It is becoming constantly more evident that even a slight departure from the optimal in the composition of the food may lead to states of nutritional instability which contribute to a physical breakdown when infectious processes are operating and the span of life may thus be shortened.

Larimore found that lesions in the esophageal portion of the stomach in rats occurred in Vitamen B, deficiency consisting of an inflammatory oedema of the mucosa and submucosa and localized ulceration. There was also an epithelial hyperplasia. In poly-neurotic pigeons the adrenals increased 2/3 times in size, although the body weight and weight of the pancreas, thyroid, liver and stomach decreased.

Lecithin is shown to have a marked influence upon nutrition. There are doubtless many other valuable compounds in this unexplored field. The mystery is locked up in the accessory factors of diet. The importance of unraveling the unknown factors in ear, nose and throat pathology wrapped up in bio-chemistry may be appreciated only too well when we consider the high percentage that fall within our domain.

Terman stated that fourteen million out of twenty million had some physical defect and that two million had some grave defect of malnutrition; eleven million are said to have enough bad teeth to seriously impair health.

My experience with soldiers overseas and the reports from the Draft would indicate that these statistics were not greatly exaggerated.

In a study of proteins, kidney, liver and milk stand out as of unusual value and among the cereal grains, wheat takes first rank and a combination of two-thirds wheat and one-third protein gave the best results in growth. In discussing the question of a high protein dietary and kidney damage, Newburg and Clarkson (1923) found that in most rabbits fed on 27% or more meat they developed nephropathy with infection due probably to the excessive secretion of certain amino acids. These views are not supported by McCollum who believes that a generous protein diet over a fair margin of safety produces the better growth and vigor in the young.

Chittenden's experiments reducing the accepted 116-120gms. of protein a day to one-half are accepted by McCollum as advantageous. A fourth Vitamin D is necessary.

If we are to have well nourished and resistant tissues in the ear, nose and throat, we must have a well balanced basal metabolism, as may be illustrated by the plus and minus findings in hyper and hypo-thyroidism and the associated vaso-motor phenomena. With the advent of elucidation in the field of bio-chemistry, the importance of calories and inaccurate chemical analysis of foodstuffs recedes and the fascinating study of the Vitamin jumps into the foreground.

The popular conception that any diet which complies with the composition of ordinary combinations affords and furnishes protein and energy in palatable and digestible form would promote growth and maintain health and long life is destined to pass into the background.

A ship of the Japanese Navy in 1884 made an eight-months cruise and many of the crew developed beri beri. A second ship made the same cruise with an improved diet and beri beri was eliminated. It was not until 1897

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that the scientific explanation was made when beri beri was produced in chickens in two to four weeks by feeding an exclusive diet of milk and rice.

It was, therefore, established that one of these was Fat Soluble Vitamin A, plentiful in butter and fat, egg-yolk, cod-liver oil, in the fats of the liver and kidney but almost absent in vegetable oils. The second Vitamin B, distributed liberally in natural food such as tomatoes, soluble in water and dilute alcohol but never found in animal or vegetable fats and oils.

Soubra found in cockerels fed without Vitamin B that there was a decrease in the size and weight of the organs in the following order—Testes, spleen, heart, liver, kidney, pancreas and thyroid. Matthews reports the addition of Vitamin B to the diet of infants suffering from metabolic disturbances increases appetite, weight and number of red blood cells. That resistance to infection is lowered is proven in the experiments of Guerrine, who fed pigeons on polished rice and found them no longer resistant to the bacillus of anthrax. Rats and guinea pigs fed on a cereal grain showed delayed wound healing and lowered resistance to other infection.

In Vitamin A deficiency, Blegoad reports a case of night blindness that failed to clear with six weeks of Vitamin feeding. He made marked improvement in 2—6 sub-cutaneous injections of one C.C. each of potent Vitamin A Solution.

Osborne and Mendel found calculi of calcium phosphate in the urinary tract of 91 animals in 857 autopsies, 43% of which had a deficiency diet of Vitamin A.

While drying of the lachyrimal gland may be one of the first pathologic changes noted, the same effect takes place in the glands of the throat and mouth and the animal may sneeze and cough violently. The mucous membrane of the larynx and trachea at autopsy shows xerotic changes. The salivary glands also are affected. In rats the secretion almost or entirely ceases. The cells on histologic examination are shrunken and irregular in size. The ducts of these glands show corrification and desquamation of the superficial cells. The sterility of rats is relieved by feeding lettuce, meat, whole wheat, wheat germ, especially the oil, rolled oats, dried alfalfa and quantities of milk-fat. The Vitamin X in this combination is classified as the fertility conferring factor. Lack of Fat Vitamin A or phosphorous leads to profound changes and loss of function in the testes of the male.

Daniels, Armstrong and Hutton in 1923 stated that rats in Vitamin A deficiency may die of microbic invasion of the ear and nasal cavities before Xerophthalmia appears. Feeding Vitamin A does not restore the condition.

Hume shows that irradiation with a Quartz Hg Lamp prolongs the growth of rats without Vitamin A, but failed to get response when they were on the diet over ninety days. Irradiation is not a substitute for Vitamin A.

In Michigan and especially the northern peninsula, thyroid enlargement is quite prevalent. According to McCarrison, thyroid hyper plasia may be readily produced by diets deficient in iodine and especially where animals live in dirty cages. His work also in 1922 brought out the direct influence of infection in the mouth, nose, tonsils and gastro-intestinal tract.

A detailed study of iodine deficiency in four counties of Michigan where 31,612 children were examined, showed 14,914 had goiters, 40.5% being boys. To remedy this iodine was recommended in salt.

Ricketts is prevented among young animals in the zoo by feeding liver, flat bones with marrow and fat and whole rabbits or pigeons. It is interesting to note that the disease is very rare in Greenland or Iceland, Norway or Sweden, or among the Eskimo. It is rare in the west of Ireland or Greece. It occurs more frequently between November and May. It is estimated by Hess and Unger that 50-75% of artificially fed children in large cities have Ricketts; 86% of those dying under eighteen months showed evidence of the disease.

A presentation of these specimens of white rats prepared in our laboratory at the Detroit College of Medicine and under the direction of Dr. Emmett, Parke-Davis & Co., confirms the findings of other observers, espe-

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cially Daniels at the University of Iowa, and demonstrates that a diet deficient in Vitamin A, which is plentiful in butter fat, egg-yolk, cod-liver oil and the fats of liver and kidney, exhibits on the 28th day of a lowered resistance to infection. It shows that these rats have a well developed nasal sinus infection and also extension to the middle ear. The controls in great numbers fail to develop suppuration. If the deficient diet is carried on later Xerophthalmia results. The importance of these findings is illustrated by observations on children reported by Mari.

Mari, in Japan, describes 1400 cases of deficiency in Vitamin A, found among children 2—5 years who were fed rice, cereals, beans, barley, vegetables. This was not found among fisher-folk and was cured with cod-liver oil, chicken livers and eel fat.

Xerophthalmia described by Livingstone in 1857 after living on sugarless coffee, roots, and meal, or by experiments on pure gluten and starch, shows the brunt of the symptoms in dryness of the conjunctiva and corneal ulceration extends to the lachrymal apparatus and the nose. Again this pathology illustrates the inseparable relationship of the eye with the nose and throat.

Bloch reports a series of fifty cases of Zerophthalmia in children fed separator skimmed milk, pasteurized and cooked again at home, together with oatmeal, gruel and barley soup. These children developed borderline scurvy, beri beri and ophthalmia in pronounced form. The cases certainly illustrate the unappreciated seriousness of malnutrition associated with tonsils and adenoids and which in many cases fall but little short of a deficiency disease.

The association of night blindness, conjunctivitis and spring time catarrh was reported by Vetsch as relieved by cod-liver oil. The relation of starvation for Vitamin A to changes in the so-called glandular adipose tissue has been noted by Cramer. This variety of tissue is exhibited by the cretins in deposits around the neck and axillae and relieved by thyroid feeding. It is suggested that the glandular adipose formations are reservoirs of Vitamin A.

The problems of hypo-thyroidism when exaggerated often show the following appearances in the nose, throat and ear. The nasal mucous membrane may be infiltrated and later thickened and the nose obstructed by a gelatinous waxy or yellowish secretion. The patient may complain of a full cramped feeling in the throat and changes of coloring to a pale yellow or bluish tinge ensue. Slight oedma with angio neurotic phenomena and a transparency of the interarytenoid fold without change of function in the vocal chords but with a dry throat or husky voice may be found.

It is recognized that the essentials in the prevention of Ricketts are Vitamin D, with a well maintained ratio of calcium and phosphorous. One pint of cow's milk gives one grain of calcium oxide; human milk contains one-fifth as much. In children, 3—13 years, it was found that the optimal storage of calcium takes place when a child has a quart of milk a day.

The role of diet to increased susceptibility is well proven. The dietary of Voegtlin showed that the addition of liver was of great value. Manifestations of deficiencies in aggravated form where a lack of phosphorous, iron, calcium are noted, may occur in the nose, throat and ear, as well as the general system. The secondary anemias, the lowered coagulation point, the poor skeletal growth and the thyroid imbalance call for a proper therapeutic selection.

In order that the hormones or regulators of metabolism may operate it is essential that iodine shall be present to the extent required.

The observation of Goldberger in a Georgia Asylum of adding fresh meat, eggs, milk and fresh vegetables in quantity while controls were kept on the old diet, demonstrated that Ricketts appears in as early as thirty-four days to three months, but usually occurs between the 6th and 24th month. Late Ricketts may appear in older children or young adults. It is of particular interest to the oto-laryngologist because the most noticeable signs are seen in the bones, and those of the head especially. These children sleep

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poorly. They may wear the hair from the back of the head rolling on the pillow. Constipation is the rule. The child does not walk. A rachitic rosary may develop where the bones of the ribs join the cartilages and pigeon-breast deformities begin. It is probable that the oto-laryngologist sees more of these onsetting symptoms with tonsils and adenoids than any other special worker except the pediatrist. The deformities of the nose, naso-pharynx and hard palate, the square shaped head, the boney formations on the side and front of the skull are developed. Curvatures take place and the jaws and epiphyses swing out of alignment.

In conclusion it may be said that while the research laboratory alone will be able to furnish a final solution to the interesting relationship that exists between food and pathology in the ear, nose and throat and elsewhere, we may well believe that a genuine practical application of dietetics and therapeutics is indicated in oto-laryngology together with our surgery. The demand of the human economy for iron, phosphorous, calcium, iodine and vitamins, is present in oto-laryngologic pathology more frequently than in other deficient parts of the body. Our role and our influence in the realm of pediatric oto-laryngology is most extensive and as malnutrition, adenitis and infection, are more prevalent in our domain. If, therefore, I have succeeded in attracting your interest and study in this direction my mission will be fulfilled.

Discussion opened by Dr. Frederick A. Keihl, Portland.

Dr. Shurly has spoken fully and as always, authoritatively, and the little I can offer is merely by way of further commending to you the importance of this subject. Particularly is Dr. Shurly to be congratulated upon the interest he has aroused among his civic authorities as manifested by their substantial appropriation in furtherance of his experimentation.

So far as I know, Kaufman, to whose work Dr. Shurly refers, first called attention to the necessity of investigating the role of vitamines in ear, nose and throat conditions, arguing that, when all other tissues are affected by diet, it is not likely that the ear, nose and throat escape. His work, first in the Beck Clinic and later at the Michael Reese Hospital, brought to notice the fact that otosclerosis—"adult rickets" as it is sometimes called—presented boney changes identical with those of the long bones in rickets, suggesting that otosclerosis may be a late result of rickets or a manifestation of a dietary deficiency still existant in adult life. Further, that hyperplastic ethmoiditis and otosclerosis present similar pathology, viz., interference with the nutrition of the bony capsule of the labyrinth in the one instance, and with that of the ethmoidal capsule in the other.

The work at Iowa University by Daniels and Armstrong and at Washington University, St. Louis, in the Department of Pediatrics, further points to the proof of the production of nasal sinusitis by a diet deficient particularly in fat soluble vitamines.

In England, Mellandy, Professor of Physiology at Sheffield, some of whose deductions Dr. Shurly has quoted, has also emphasized the similarity between faulty dental development, rickets and catarrhal infections of the respiratory tract. Female dogs were given during pregnancy a lowered diet, such as if given puppies would insure the development of rickets and similar mal-nutritive conditions. It was found that the offspring of such under-fed bitches were particularly prone to develop the same conditions. His conclusion then is that it is fair to assume that the so-called "catarrhal child"—i.e., the child subject to chronic catarrh of the respiratory tract, with tendency to broncho-pneumonia and diarrhea—this state often appearing in the first few weeks of life before post-natal conditions could be held accountable—that such cases may well be explained by defective maternal diet. Again, we must start with the grandmother or earlier to breed for health.

It has been known for thirty years that improper diet causes "snuffles" in hogs and we are only now giving children the benefit of this knowledge. It is our duty to combat all fads in feeding, and to do all we can towards

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the education of mothers as to the importance of all food factors in proper proportions that they may avoid for their children the many metabolic disorders that spring from unbalanced dietaries.

Little is yet known about the actual transfer of food elements to the cells of the body. "When this physiological problem is better understood we shall be better able to recognize early, slight deviations from the normal which are later to result in some deficiency disease."

Dr. Wilson Johnson, Portland, Ore.:

I want to thank Dr. Shurly for giving me a scientific justification for my unexplained antagonism to angel cake. Looking back over the literature, we find that clinicians have for years been seeking for something in the way of a dietary that would have to do with particularly cell reconstruction, and it is only in the last few years that we have realized the importance of vitamins in this process. The work of McCollum is mentioned and the work of Whipple has gone far to give us, not only recognition of vitamins, but also to specify the vitamin that is responsible for the action. Whipple has also recognized the use of liver and kidney.

I am reminded of an incident I had forgotten. I had a friend, a medical student, a very decent chap, in New York in 1896 and the lad told me this: He said he never had been in the habit of drinking beer. He didn't like it, but after he had been in New York about three or four months every once in a while he would have an irresistible desire for beer. That habit grew until he had to have it every day. His father was an old country physician and he wrote his father, and his father wrote back: "Son, your diet is insufficient; if you will eat raw onions and raw cabbage you won't need the beer."

One question I would like to ask Dr. Shurly. Some of us have felt that the administration of calcium should not be made in the presence of an excess of fats, particularly animal fats, etc.

Dr. Fred T. Hyde, Port Angeles:

In cases of tonsillectomy following the operation varying from three months to a year, the complaining child has frequent colds, etc., maybe earache or return of deafness. You look into throat and are surprised to find lymphoid tissue, particularly behind posterior pillars. Sometimes as much as tissue removed at operation. The mother is not satisfied and neither are you. If Dr. Shurly or any other gentleman can inform me of the cause of this increase and how we can get away from it I will feel very much gratified.

Dr. Burt R. Shurly (Conclusion):

Answering Dr. Johnson's question about administration of calcium in the presence of excess fats, particularly animal fats, I have been unable to find anything in the literature or in any laboratory experience which we have had that shows any special contra-indication to the administration of calcium in the presence of excess fat of any variety.

Answering Dr. Hyde's question about recurrence of adenoid tissue, the problem of recurring adenoids is undoubtedly unsolved. Theoretically, of course, the wild or regular growth of cells is wrapped up in this problem of bio-chemistry with its intricate problem of cell selection. The electrical problem of the cell, the problem of its action under irritation, is constantly before us in the form of polypi, papilloma, and also the cancer cells. The islands of Waldeyer's ring have circulatory vaso-motor problems in common; varieties of tissue must have phenomena of cell selection that differ. Our experimental information about Vitamin B, its affect upon the growth of lymphoid tissue may in a crude way be a part of the explanation. It is a curious fact of nature that when something is removed, she tries to restore and repair the process as far as possible. Recurring infection no doubt is also stimulation to the cell growth.

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FRONTAL SINUS EMPYEMA IN YOUNG CHILDREN WITH SEVERAL CASE REPORTS *

IRVING MARTIN LUPTON, M.D., Portland, Oregon

It is more or less the consensus of opinion that frontal sinuses of sufficient size to be considered as a surgical entity, occurring in young children, are an exception to the rule, and are not commonly found. This is true, but nevetherless we must not allow this fact to overrule our judgment and discard the possibility because of its more or less rare occurrence.

Hajek states that the development of the frontal sinus does not begin until the first year of life and it reaches the size of a bean at the sixth to seventh year of life. It does not reach its permanent size until the complete development of the body.

Davis states that as an average the frontal sinus is about 3.8 mm. above the nasion at three years of age and an advancement occurs of about 1.5 mm. a year until the fifteenth year. This would appear at variance with the radiographic findings in which so many absent frontal sinuses are recorded. Considering the small amount of space involved it is possible that a frontal sinus may be confused with and charted as an ethmoid cell, thereby increasing the number of frontal sinuses tabulated as absent. Radiographic findings are an interpretation of shadows, while on the other hand the measurements are actual findings.

According to good authorities, the frontal sinus has a clinical significance at the age of five. Killian reports operation on the frontal sinus of a child fifteen months old. E. Myers reports operation on a child three and one-half years of age. Dean states that the anatomic knowledge of the average size which any sinus should have at a certain age is not important considering the fact that there may be a precocious development of any sinus so that it might be a surgical sinus much sooner than one would expect.

We have in our experience been surprised by the fact that in the routine study of radiographs of young children a marked disparity exists between the acutal findings and the prevalent belief that the frontals do not exist at this early age; or, may I say, are not of sufficient size to be considered as an entity of surgical importance.

In several instances, much to our dismay, this attitude has dominated or influenced the diagnosis and the possibility of frontal sinus disease has been discarded as most improbable, and eliminated as a factor to be considered in the diagnosis of the little patient.

In many cases seen a diagnosis is made of abscess within the confines of the orbital cavity, most likely as a result of an extension of active disease in the ethmoidal cells. We have in several instances incised what was supposed to have been such a condition and have ended by opening one or both frontal sinuses, which were of great size, containing thick mucosa, polypoid in character, and the sinus literally filled with pus. This was despite the fact that the radiographic findings were more or less indefinite as to the presence of an actual empyema. There is also that type of case in which frontal sinusitis has been unrecognized and a fistula may have resulted simulating a dacryocystitis. It is logical to assume that in time past many so-called cases of ethmoidal empyema with orbital extension have in reality been complicated by severe frontal involvement with all its attendant dangers.

Confusion necessarily must arise when a dense shadow over the frontal region may be due to so many conditions, among which I might enumerate sinusitis and empyema—a shallow cavity or thickened lining membrane—periostitis—luetic bone or osteomyelitis, or an absence of a sinus. Often the presence of a hard solid oedema overlying the sinus and filling the upper portion of the orbital cavity may confuse sharp detail outline, giving rise

* Dr. Lupton, having been delayed, Dr. Mellinger read paper.

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to doubt regarding involvement. A shadow shown in the shallow frontal or the thickened plate of bone over the sinus in many instances is very suggestive of a pathological involvement. Any condition affecting the anterior sinus wall such as a periosteitis, osteomyelitis, luetic or tuberculous involvement, will of course simulate sinus disease. To differentiate it is necessary to have a lateral view.

A study has been made of many radiographs in file at the Doernbecher Memorial Hospital for Children at the Shriners Hospital for Crippled Children and in private collections, not only our own, but those of several of our colleagues. In approximately 11% of over two hundred films picked at random the frontals were easily distinguished and were of greater size than generally conceded for the age of the child. Not a few of them approached adult dimensions. According to Skillern the frontal sinus embryologically is merely an offshoot from the ethmoid labyrinth. In the disarticulated frontal bone the frontal sinus appears merely as a cavity divided by a septum. The ethmoid closes in these apertures from below, thereby forming the floor of the frontal sinus. It will be seen that the floor of the frontal sinus is in reality a portion of the ethmoid capsule. This fact may explain why frontal infection is easily possible when an overdeveloped sinus is found. It also probably explains why infection is so easily extended upward secondary to acute ethmoidal involvements, due to the incomplete evolution of the anterior ethmoid cell into the frontal sinus proper.

The anterior wall of the frontal sinus is composed of cancellated bone and varies in thickness from one-sixteenth of an inch upwards. The posterior wall is thinner, rarely exceeding one-sixteenth of an inch in thickness even in the adult. This structure forms part of the wall of the anterior cranial fossa and is in contact with the frontal lobe of the brain. Defects in these walls have been found. On account of the thin posterior wall, such possible defects must be considered in relation to inflammatory processes. This same condition has long been known in regard to involvement of the mastoid in children without apparent otitis media and has received special consideration from pediatricians and otologists in the last few years.

It seems probable, as pointed out by Oppenheimer, that many cases of meningitis are the result of some unrecognized inflammatory condition of some of the sinuses, notably the frontals and sphenoids. The bony walls of the sinuses in children are much softer and cartilaginous than found in the adult. The mucosa in children is in more intimate relation with the osseous tissue so that the slight inflammation of the mucosa must often produce some pathologic change in the bony walls of the sinus. The lymphatic and vascular systems show a more profuse development in children than is found in the adult.

From our own records we have selected three rather typical cases illustrating the various points in the question, the rapidity of involvement and the attendant dangerous possibilities.

Case 1—Acute left ethmoiditis; orbital cellulitis; double frontal sinusitis; external radical frontal and ethmoid operation; exposure of dura; recovery. A. H. white boy, 11, robust, admitted to Multnomah Hospital December, 1921. This boy had been suffering from pains in his face and over his eyes following diving during the preceding summer. He had been sick for two months, and a swelling over the left eye had been opened by a neighbor, four months before admission. Yellow streptococcic pus discharged from the fistula since. Temperature was 97.8, pulse 74, white count 9500; Radiograph showed dark left frontal, with no apparent right frontal involvement.

Radical external left ethmoid operation December 15, 1921. There was little posterior involvement, but a rather large left frontal, placed almost horizontally, with no extension upward into the frontal bone, was found to communicate directly with the right frontal. The partition between was loose, coming away as a sequestrum. An area of dura 1 by 2 cm. was exposed above the left frontal.

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Postoperative temperature ran from 97 to normal. There was no diplopia, and the boy left the hospital nine days postoperative and has been well since his discharge from office care a few weeks later.

Case 2—Right ethmoiditis; orbital cellulitis; right frontal sinusitis with meningeal extension; external radical ethmoid and frontal operation; immediate recovery with subsequent death from other causes.

C. K., white boy, 11, admitted to Portland Ear, Nose and Throat Hospital September 24, 1924.

Following severe head cold developed severe pain in region of right eye. During course of next few days pain became generalized over head, accompanied by septic type temperature chills and sweats. Convulsion occurred on day admitted to hospital. Pain became localized over region of right frontal sinus. W.B.C. 22000. Right nasal passage filled with pus. There was a definite orbital cellulitis present. Patient restless. Radiographs revealed dark right ethmoid and frontal sinuses. The right frontal appeared to be of great size. Left frontal small and clear.

External ethmoid and frontal operation performed. The right frontal sinus approached adult size, measuring approximately 3½ cm. in its lateral diameter and extending upward into the frontal bone 2½ cm. It also encroached over to the left of the mid-line. The dura over the right frontal lobe was exposed during the operation and there existed a localized meningitis with epidural abscess formation. Extension and perforation occurred through the lamina papyracea, explaining the orbital involvement.

Post operative progress complicated by septic pneumonia, endocarditis and an acute hemorrhagic nephritis. Patient left hospital in four weeks with temperature normal and wound closed. Absolute rest was carried out at home for the following month and patient up part time. The renal complication, however, persisted and death occurred five months post-operative. Autopsy refused. Hemolytic strepticocci culture.

Case 3—Right acute ethmoiditis; orbital cellulitis; bilateral frontal sinusitis; external ethmoid and frontal operation; recovery.

A. C., white boy, 9, admitted to Portland Eye, Ear, Nose and Throat Hospital October 14, 1926. Several days following swimming and diving lesson developed severe head cold. Several days following, an oedematous mass appeared in the mid line of the forehead directly above the nasion. Severe frontal headache was complained of at this time. Moderate temperature. The attending surgeon incised the mass, but obtained no pus. Relief from headache was however obtained for one day with reappearance of violent headache. The headache increased in severity for 48 hours, then suddenly disappeared and the child was apparently well, other than the swollen forehead. Within 24 hours, however, the headache again appeared to remain for one day and with its cessation complete inability to breath through the nose, which heretofore had not been complained of.

At this time we were called in to see the case and the findings were those of septal abscess with complete nasal obstruction. Temperature 101. W.B.C. 20000. An oedematous mass presented in mid line of forehead, size of half an orange.

Radiographs showed dark right ethmoid and frontal. The left frontal sinus did not appear to be involved to any great extent. A dark shadow extended down the mid line of the nose corresponding to the line of the nasal septum.

External right ethmoid operation performed. The ethmoid was filled with pus. The right frontal sinus measured 2 cm. in its lateral diameter and 2½ cm. from the floor to its greatest heighth. A perforation had occurred throught the anterior wall of the right frontal connecting with a large subperiosteal abscess. A perforation had also occurred through the floor of the right frontal into the orbital cavity. The partition between the right and left frontal was partially destroyed and the left frontal, though small, was filled with pus. Pus had also burrowed down along either side

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of the septum lifting the muco-periosteal covering from the bony-cartilaginous nasal septum, ballooning them out and causing a complete nasal obstruction. Free incision was made through the anterior nasal opening into the most dependent portion of this septal extension.

Postoperative temperature ran from 103 to normal. Patient left hospital in ten days and has been well since discharge from office treatment.

An interesting feature that we have observed in several cases is associated maxillary sinus involvement on the same side as the frontal infection. Clinical findings and symptoms may point only to the maxillary sinus. Treatment directed to the antrum alone does not produce the desired result. In all maxillary infections which do not properly respond, special effort is made to rule out an associated frontal infection.

The disease in children runs a very rapid course when once established and often most radical procedures must be followed to alleviate the condition, not to speak of saving the child's life. Coffin has flatly expressed the opinion that there must exist some period during the course of a frontal sinusitis in which proper treatment would have prevented most serious complications.

Consideration of the treatment of frontal infection in children must ever be guided by the effort to preserve essential structures. Constant vigilance must be exercised during any acute nasal infection in children against the ever present possibility of frontal sinus invasion. Before we can expect any permanent relief to frontal sinus infection attention must be directed to the anterior group of ethmoid cells, as their relation to the frontal sinus is most intimate. The employment of some type of suction treatment for freeing the nose and ostia of secretions will always be of great value. Infraction of the middle turbinate on the affected side followed by thorough shrinking and the application of gentle suction has been of great value in our hands in beginning invasion. The use of ephedrine 1½ to 3% sprayed or dropped in the nose at very frequent intervals has facilitated drainage and aeration of the sinuses, and has proved a most valuable adjunct, lacking the deleterious effects of adrenalin or cocain. Removal of the anterior tip of the middle turbinate is sometimes necessary, especially when that structure is bulbous and obstructive. This procedure is usually done as a preliminary step to the external frontal operation. Intranasal operations alone are of little avail if much oedema and redness has occurred externally in the region of the eye. We should be most reluctant to do the external operation in the absence of either external signs or intracranial symptoms. We are inclined to be rather conservative even in cases where the external operation is found to be necessary. No attempt has been made on our part completely to remove the anterior wall of the sinus, as is done in the adult case. Merely enough of an opening is made to facilitate clear vision and to give us a reasonable amount of space in which to do the necessary work.

A thorough exenteration of the anterior group of ethmoidal cells gives us the necessary approach to the floor of the frontal sinus. This is opened from below and enough of the floor is removed to assure us that proper drainage will be established. We consider that by this procedure that we increase our factor of safety.

We have seen two cases of generalized osteomyelitis of the frontal bone following the frontal operation in children, and these had followed most conservative exploration through the anterior sinus wall. This same frequency of osteomyelitis following large resection of the frontal plate has been adversely commented upon by Geo. Swift in connection with his work in surgery of the frontal lobe.

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Discussion opened by Dr. Jones, Boise.

My experience with these cases is rather limited. Have not had any children under 15.

Dr. Frank R. Spencer, Boulder, Colo.:

Dean Advocates the use of 3% Ephedrin Sulphate followed by 4% Argyrol. He says that he can cure most of the cases of sinusitis in children by this method of treatment, providing the pediatrician is not in too great a hurry to have the foci of infection in the sinsus eliminated. Doctor Carmody, who is here, and whose "plunder" I may be stealing, has mentioned the fact that the frontal sinuses often do not develop in children if the ethmoidal labyrinth has been infected during the developmental period.

Dr. *Ira E. Gaston,* Portland, Ore.:

I wish to express my appreciation of this very excellent paper. Of course the cases described represent severe types of infection. We see cases quite frequently in children of orbital cellulitis from sinusitis which clear up without surgical intervention. Even sub-periosteal abscess from frontal sinusitis may not require surgery of the sinus. A case in point is that of a child, age 13, who developed a sub-periosteal abscess under the supra orbital ridge during the course of an acute pansinusitis. Incision of the abscess and evacuation of the pus revealed no demonstrable fistula leading into the frontal sinus. Since the acute symptoms were subsiding, the sinus was not opened. The patient made a rapid and complete recovery from the sinus infection.

Dr. Wm. Mellinger, Santa Barbara:

I would like to ask Dr. Carmody a question. Whether or not the affect of the lack of air passing through the nose is as great a factor as is the infection, upon the development of the sinuses. That is in a case of nasal obstruction without sinus involvement.

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PYOCYANIC STOMATITIS WITH AGRANULOCYTIC LEUKOPENIA

F. H. LINTHICUM, M. D., Los Angeles, Calif.

In no text-book devoted to diseases of the throat has it been possible to find a description of a stomatitis associated with the bacillus pyocyaneus, and it is because it may fall to the lot of any ear, nose and throat specialist to encounter such a condition, that the following report is offered for your consideration.

In 1899 Geoghiewsky claimed to have found a leucocyte-destroying ferment in cultures of the bacillus pyocyaneus.

In 1924 Lovett reported a fatal case of ulcerative stomatitis associated with extreme leukopenia in which the bacillus pyocyaneus was associated as the apparent cause. This was the first case described as occurring in this country. During the year prior to this, however, about a dozen cases had been reported in Germany, and in two of these pyocyaneus was found. The syndrome is variously known as "Agranulocytic Angina," "Agranulocytosis," or as suggested in the present title, "Pyocyanic Stomatitis with Agranulocytic Leukopenia." This name is suggested because, as will be explained later in detail, we have had occasion recently to see a case of ulcerative stomatitis accompanied by an extreme leukopenia, in which the bacillus pyocyaneus was not identified and in which the patient recovered.

Both in the case to be reported in detail today and a second one to be mentioned, the bacillus pyocyaneus was recovered in pure culture. Both of these cases terminated fatally.

All the cases of the disease reported from Germany in 1923 terminated fatally. However, in only two of them, as in the one reported by Lovett in this country in 1924, was the bacillus pyocyaneus identified. There have been probably half a dozen instances of Agranulocytic Angina reported in American literature since Lovett's case, but in none of them has the organism in question been found, until in the present two instances:

The first patient in question was a woman aged 42. Had been married twelve years. Had been delivered of her first child by Dr. Morris Simons by caesarian section five weeks prior to the onset of her present illness. Pregnancy had been complicated by a pre-eclamptic toxemia. She had been in bed since the delivery of her child, and when I first saw her, she was complaining of a sore throat which had developed the previous night. Her temperature was 100°; her pulse 90. There was a small grayish patch on the right tonsil with no involvement of the adjacent lymph glands. The next day the temperature was 101.4°. The right tonsil was clear, but the left one had become involved in a manner similar to the opposite side. The tonsillar condition then apparently cleared up. During the next few days an exudate formed along the gingival margins. It spread to the inner aspect of the cheeks; the floor of the mouth and the ventral surface of the tongue. It did not extend to the tonsils or any other structure outside the buccal cavity. At no time during the course of the disease was there any enlargement of the cervical glands.

The exudate was thick, gray, and formed fibrinous-like bands extending from the gums to the inner aspects of the cheeks. It stripped off in large pieces, exposing a surface which did not bleed. The gum-tissue between the teeth became necrotic and sloughing. On the fifty day the temperature was 104°. The upper lip became edematous and tremendously swollen, followed shortly by a similar condition of the lower lip. By the seventh day the temperature was lower, the swelling of the lips was subsiding and the exudate in the mouth was disappearing. On the tenth day she suddenly became worse; pulse poor, dyspnoeic and very apprehensive. She began to have involuntary stools. She became comatose and died on the eleventh day of her illness.

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Autopsy was of little or no help in furnishing additional information as to exactly what type of infection had caused her death. There were ulcerative lesions in the mouth and a few on the genetalia. There was a moderate icterus. The heart was one-third larger than normal and on histologic examination showed acute degenerative changes. The liver and kidneys showed evidence of old and acute infection. There was a healed duodenal ulcer and evidences of previous tuberculosis. There were other findings which had no bearing on the terminal illness, except to indicate that we had been dealing with an individual whose powers of resistance were generally below par. Immediate cause for death was given as an ulcerative stomatitis and acute myocarditis.

The character and distribution of the exudate; the absence of glandular involvement; the course of the disease, suggested neither diphtheria nor an infection with Vincent's organism. Nevertheless, since spirillae and fusiform bacilli had been found in moderate numbers, it was deemed advisable to treat the case as one of Vincent's Angina. Salvarsan was administered intra-venously and applied topically, and the gums and mouth received suitable hygiene.

A day or so after the patient's death a re-examination of a portion of the membrane obtained from the mouth during the early stages of the disease revealed the presence of numerous bipolar staining organisms which were thought to be those of diphtheria. These were soon found, however, to be actively motile and gram negative and were finally identified as being the bacillus pyocyaneus. This resemblance to the diphtheria bacillus has been mentioned by other workers, notably Park in his text-book on Bacteriology. Gram's method of staining, however, will differentiate these two organisms since pyocyaneus is gram-negative and the diphtheria bacillus is gram-positive. Pyocyaneus is easily cultivated in both aerobic and anaerobic cultures and produces a characteristic pigment on suitable media.

Since the original report of a case of this kind in this country in 1924, there have appeared at various times in the literature, descriptions of instances of the disease known as Agranulocytic Angina. The bacillus pyocyaneus has not been found in these cases.

E. C. Piatt, in a report on the histo-pathology of Agranulocytic Angina, discovers in the lesions found in the disease, a gram-negative bacillus of which he has to say: "If they belong to the bacillus pyocyaneus species, they must possess some peculiarities in their biologic properties." He furthermore concludes that the ulcerative areas in the mouth, vagina, spleen, etc., are but "secondary lesions as a consequence of the septic pyemia."

Henry S. Weider reports a case in which the patient suffered two attacks, the second one fatal, and in which the bacillus pyocyaneus was not mentioned.

R. J. Hunter in 1926 reports a case following a fractured tibia. He reports cultures from the lesions as showing only the usual flora of the mouth.

Harold P. Hill of San Francisco has recently reported a case following the extraction of teeth, in which the bacillus pyocyaneus was sought for and not found, but in which, on account of the blood picture, the diagnosis of Agranulocytic Angina was made.

Recently we have had occasion to see a case similar to the last one mentioned. Following the extraction of a number of teeth in a middle aged woman, she developed a gangrenous process in the mouth and nose. The total white blood count was 1400, with 3% of granular cells. The blood picture persisted throughout the febrile course of the illness which lasted two weeks. The white count gradually resumed its normal proportion and number. Repeated smears from the mouth and nose failed to reveal the presence of the bacillus pyocyaneus. The woman recovered.

On the contrary, Doctors Hammack and Zeiler have recently had occasion to follow still another case of ulcerative stomatitis in a middle aged woman, in which the white blood count was 2400 with 19% of granular cells and in which the bacillus pyocyaneus was recovered in pure culture from the mouth lesions. The woman died.

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Clinically this last case and the one described in detail earlier, seem to fall in a definite group, identical with the one reported by Lovett in this country in 1924, and two found in the German literature. These resemble the cases of Agranulocytic Anginas described by Piatt, Weider, Hunter, Hill, and others, insofar as the blood picture and mouth lesions are concerned, but differ from them in the important details that so far it has always occurred in middle aged women; it is apparently associated with the bacillus pyocyaneus and it invariably terminates fatally.

It would seem, then, that one must distinguish between two manifestations of the same disease. Agranulocytosis or Agranulocytic Angina may occur with any severe stomatitis; it occasionally occurs in men. It has no characteristic bacteriology. There is a leukopenia with diminution in the granular elements of the blood. Such cases are not necessarily fatal. Into this group fall all the so-called Agranulocytic Anginas.

As a subdivision of this class and one which seems to present more or less of a clinical entity is suggested that group of cases, to date five in number, which apparently occur only in middle-aged women; which in the cases so far reported, have all proved fatal and in which the bacillus pyocyaneus has been recovered in pure culture from the typical lesions or internal organs. For this particular picture is suggested the name "Pyocyanic Stomatitis with Agranulocytic Leukopenia."

Through the courtesy of the Clinical Laboratory of Doctors Brem, Zeiler and Hammack it has been possible to observe the action of the organism on guinea pigs. A culture was made of pyocyaneus obtained from the mouth of the first patient reported and varying amounts injected into the guinea pigs. In order to compare the results obtained another organism was chosen which can be either pathogenic or non-pathogenic. For this purpose B. Coli Communis was selected.

The inferences to be drawn from these experiments are:

1. That certain organisms, when injected in sufficient amounts into guinea pigs, may produce a leukopenia with destruction of the granular elements of the blood without necessarily proving fatal to the animal. These experiments are apparently corroborated by the cases occurring in man of so-called agranuolcytic angina of unknown itaology and are not necessarily fatal.

2. That under certain conditions the bacillus Pyocyaneus may become virulent and that such stains when injected into guinea pigs are fatal and have the power of producing a leukopenia and a destruction of the granular elements of the blood. Furthermore, that this organism when virulent may produce the same picture in man and give rise to the disease for which the name Pyocyanic Stomatitis with Agranulocytis Leukopenia is suggested.

NUMBER 1

CONTROL PIG

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles	Total count
May 16, 1927—10:00 A. M.	15	85				13,000
12:00 A. M.	53	44	2	1		
4:00 P. M.	51	46		3		6,900
May 17, 1927—10:00 A. M.	28	68	2	2		12,700
12:00 A. M.	35	62		3		
2:00 P. M.	32	61		7		7,900
4:00 P. M.	41	51		8		

Control showing variation in differential and total white count

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NUMBER 2

Control Pig

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles	Total count
May 16, 1927—10:00 A. M.	36	63		1		7,100
12:00 A. M.	59	34		7		
2:00 P. M.	66	27		7		18,000
4:00 P. M.	60	35		5		5,200
May 17, 1927—10:00 A. M.	53	34	3	10		13,900
12:00 A. M.	33	54		13		
2:00 P. M.	36	48		16		7,400
4:00 P. M.	30	54	1	15		

Control showing variation in differential and total white count

NUMBER 3

September 2, 1926

Pig inoculated with ½ agar slant growth Bacillus Pyocyaneus

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles
Before inoculation	42	47	1	
2 hours after	79	18	1	2
4 hours after	33	66	1	
6 hours after	77	18	1	4
22 hours after	22	77	1	
26 hours after	40	58	1	1
30 hours after	22	75	3	
45 hours after	12	87	1	
52 hours after	11	86	3	

Pig died over week-end

Pig autopsy I.

Initial leucocytosis followed by moderate leucopia with relative lymphocytosis.

NUMBER 4

September 2, 1926

Pig inoculated with 1/5 agar slant growth Bacillus Pyocyaneus

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Basophiles
Before inoculation	44	56		
2 hours after	60	40		
4 hours after	79	21		
6 hours after	91	8	1	
22 hours after	68	32		
26 hours after	59	40		
30 hours after	69	30	1	
45 hours after	58	40	2	
52 hours after	67	29	4	Marked Leucocytosis
Week later	73	25	2	Slight Leucocytosis

Pig autopsy II.

Sublethal dose showing development of leucocytosis instead of leucopenia

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NUMBER 5

SEPTEMBER 13, 1926

Pig inoculated with ½ agar slant growth Bacillus Pyocyaneus

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles
Before inoculation..	38.5	59.5		2	
2 hours after.....	53.5	45	0.5	0.5	0.5
4 hours after.....	60.5	37		1	1.5
6 hours after.....	57	39		2	2
24 hours after.....	23	72	3		2
27 hours after.....	17	82	1		Slight Leucopenia
30 hours after.....	16	82	1		1 Marked Leucopenia
32 hours after.....	17	80	3		Marked Leucopenia
48 hours after.....	7	87	4		2 Marked Leucopenia
51 hours after.....	8	86	3		3 Marked Leucopenia
56 huors after.....	13	84	3		Marked Leucopenia
74 hours after.....	1	95			4 Marked Leucopenia
78 hours after.....	4	96			Marked Leucopenia

Pig died few hours later

Pig autopsy III.

Almost complete agranulocytosis with moderate dose while organism still virulent

NUMBER 6

September 20, 1926

Pig inoculated with ½ agar slant growth Bacillus Pyocyaneus

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles
Before inoculation....	30	66	1	2	1 Leucocytosis
2 hrs. after..	62	37			1 Marked Leucocytosis
4 hrs. after..	47	52			1 Marked Leucocytosis
6 hrs. after..	59	38		1	2 Marked Leucocytosis

Pig died during night.

Pig autopsy IV.

Animal died before leucopenia had developed.

NUMBER 7

September 22, 1926

Pig inoculated with ½ agar slant growth Bacillus Pyocyaneus

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles
Before inoculation....	35	63		2	
2 hrs. after..	69	31			
4 " " .	56	42		2	
6 " " .	60	40			
8 " " .	67	33			
24 " " .	6	92	1		1 Marked Leucopenia
27 " " .	6	94			Marked Leucopenia
29 " " .	10	90			Marked Leucopenia

Pig died 3 hours later.

Pig autopsy V.

Organism recovered in pure culture from throat of animal after subcutaneous injection.

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NUMBER 8

November 29, 1926

Tan pig inoculated with 1 agar slant growth Bacillus Pyocyaneus

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles	
Before inoculation.....	41	58	0.5	0.5		Leucocytosis ? Leucocyte ct.
3 hrs. after.....	35	64	0	0	1	lower than before inoc. Total ct.
6 " "	56	44	0	0	0	higher than last count.
9 " "	65	33	2	0	0	same
23 " "	28	72	0	0	0	Leucopenia
26 " "	24	75	1	0	0	"
30 " "	22	77	1	0	0	"

Pig died during night.
Pig autopsy VI.

Culture two months older than when last used—shows decrease in ability to produce agranulocytosis.

NUMBER 9

December 13, 1926

Black pig inoculated with 1 slant pyocyaneus

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles	Total Count.
Before inoculation.....	37	49	11	2	1	20,600
4 hrs. after.....	66	32	2	0	0	22,700
8 "	64	35	1	0	0	11,300
24 " "	43	56	1	0	0	7,100
28 " "	21	78	1			5,200
32 " "	22	78	0	0	0	5,000

Pig died during night of December 14

Relation of differential to total count. Showing leucopenia with relative leukocytosis, using attenuated culture.

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NUMBER 1

September 13, 1926

Pig inoculated with ½ slant B.coli communis

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles
Before inoculation	48	51	1		
2 hrs. after	75	24	1		
4 " "	61	39	1		
6 " "	67	31.5	1		0.5
24 " "	29	68	3		
27 " "	43	55	2		
30 " "	52	45	2	1	
32 " "	60	39	1		
48 " "	43	53.5	2	1	0.5
51 " "	40	56	4		
56 " "	60	38	1		1
74 " "	32	65		1	1
78 " "	53	45	2		
96 " "	56	40	3	1	
120 " "	30	69	1		
149 " "	59	38	2	1	
173 " "	38	59	2	1	
197 " "	47	52		1	
221 " "	52	48			
245 " "	50	49		1	
269 " "	56	43		1	

Moderate leucocytosis with small dose of B. Coli Communis.

NUMBER 2

December 13, 1926

White Pig inoculated with 1 slant of communis

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles	Total ct.
Before inoculation	49	42	8			17,300
4 hrs. after	79	21	0	0	0	22,200
8 " "	70	29	1	0	0	12,600
24 " "	46	43	1	0	0	19,500
28 " "	44	54	2			14,000
32 " "	62	36	2	0	0	21,600
48 " "	61	37	1	1	0	21,000
53 " "	61	35	3	1		17,400
57 " "	69	29	1	1		17,500
72 " "	49	51	0	0		14,800
80 " "	43	53	1	3		11,500
96 " "	43	53	1	1	2	11,800
120 " "	25	65	5	5		10,200

Pig in good health

Leucocytosis following larger dose.

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NUMBER 3
September 20, 1926
Pig inoculated with growth 1 agar slant B. Coli Communis

Before	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles	
inoculation	34	61	1	4		
2 hrs. after	63	35	1	1		Leucocytosis
4 " "	59	35	3	1	2	Leucocytosis
6 " "	49	50	1			
24 " "	25	74		1		
28 " "	33	64	2	1		
31 " "	56	39	3	2		
47 " "	31	66	3			
50 " "	37	59	1	3		
53 " "	46	51	3			
71 " "	22	71	3	4		
78 " "	37	59	1	3		
95 " "	20	70	5	4	1	
100 " "	41	56	2	1		

NUMBER 4
September 27, 1926
Pig inoculated with 2 agar slants of B. Coli Communis

Before	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles	
inoculation	26	71		1	2	
4 hrs. after	15	85				
8 " "	57	41	1		1	
24 " "	14	83	2		1	
28 " "	18	81	1			Leucopenia
32 " "	21	78	1			Leucopenia
50 " "	13	83	1		3	Leucopenia
56 " "	15	85				Leucopenia
73 " "	5	84	11			Leucopenia
80 " "	5	94	1			Leucopenia
97 " "	13	87				Leucopenia
122 " "	15	83	1	1		Leucopenia

Marked leukopenia following large dose; animal recovered.

NUMBER 5
B. Communis
November 29, 1926
Brown speckled pig inoculated with whole slant communis

Before	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles	
inoculation	68	29	2.5	0.5		
3 hrs. after	76	22	1	0	1	
6 " "	86	14	0	0	0	
9 " "	77	24			1	
23 " "	60	37	1	2		
26 " "	46	52	0	1	1	Leucopenia
30 " "	31	65	1	0	3	Leucopenia
48 " "	7	92	1	0	0	Leucopenia
52 " "	10	89	1	0	0	Leucopenia
56 " "	12	79	0	0	9	Leucopenia

Pig died during night of December 1, 1926
Pig autopsy VII
Marked leukopenia with death.

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7. Note: All laboratory work has been done through the courtesy and in the clinical laboratory of Doctors Brem, Zeiler and Hammock.

Discussion opened by Dr. Peter A. Jordan, San Jose, Calif.:

I have been instructed by Dr. Linthicum's interesting paper. Fortunately I have never knowingly had such a patient. To me it resolves itself into the following:

I may be called upon to treat a case of angry stomatitis, with a leucopoemia. The patient may have an ulcerative stomatitis with an exudate which peels off without bleeding. The white count may be as low as 1400 where it normally would be at 8000. Laboratory findings prove the absence of diphtheria. Yet our prognosis is in doubt. The anxious family cannot yet be assured. If B. Pyocyaneus is absent the patient will recover. If, though, in exactly the same clinical picture B. Pyocyaneus is lurking in the hidden recesses of cells or intracellular structure, then the patient will die.

Manifestly, then, I must urge my technician to quickly isolate a bi-polar organism, actively motile, and gram-negative. Then I have at least a diagnosis. But it spells death. If the organism cannot be found our diagnosis is in doubt until the patient proves the absence of B. Pyocyaneus by recovering, proves the presence by dying.

Our only hope is in a more effective treatment. Dr. Flehme of Spokane tells me that a vaccine treatment is best.

Dr. Hyde, Port Angeles (Pyocyanic Stomatitis):

It is very highly probable that a great many cases we have had which unfortunately have passed away and which we thought were Stomatitis, might probably have been a focal infection unrecognized simply because we did not look for the Bacillus Pyocyaneus.

Three weeks ago I saw a young man in the hospital who five weeks previous had been operated on for a ruptured appendix. He developed a distinctly foul looking membrane in nose and throat not diphtheria. Did not respond to treatment whatsoever. Three days after developing membrane he died. He had distinctly low resistance from appendectomy but the membrane was one I had never seen before and truly unusual in appearance, and I think it was the straw that broke the camel's back, etc. I think it highly probable that this was a case of Pyocyanic Stomatitis.

Dr. A. T. Wanamaker, Seattle:

As I listened to the paper the question came to my mind: "Has alcohol or diluted acetic acid been used in the mouth?" We know these agents are most helpful in the ear for pyocyanic infection.

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TRICHLORACETIC ACID IN THE TREATMENT OF ULCERATIVE LARYNGEAL TUBERCULOSIS

BERTRAM C. DAVIES, M. D., Los Angeles, Calif.

In presenting this report of a new treatment for tuberculous ulceration of the larynx, a preliminary review of the multitude of therapies to which this affection has, in the past, been subjected, has been purposely omitted, as well as a discussion of the percentage of patients so affected. This is simply a record of the results of this particular method of treatment. During a period of ten months, 86 cases were seen, ranging in age from 14 to 62 years.

Dysphagia is often the barrier which prevents the recovery of tuberculous patients. Such individuals must be fed, but when swallowing is accompanied by intolerable pain, or made impossible because of an ulceration in or about the larynx, they refuse food and the fight is lost.

A review of the literature carries one through the gamut of such forms of therapy as inhalations, sprays, insufflations, pigments, injections and surgery. All of the different methods have had their champions for a time. Some have apparently produced favorable results under certain conditions. As a rule, however, they are too slow in their action, resulting in the patient losing ground too fast, waiting for the dysphagia (odyuphagia) to be overcome, and dying from malnutrition.

During the past two years the writer has been using Acid Trichloracetic Pure in the treatment of Vincent's Angina. In this disease it was found to give the best results in producing a clean field with rapid granulation. One year ago this treatment was used on a patient sixty-four years of age affected with a tuberculous ulceration on the posterior surface of the epiglottis. The patient was much emaciated because of the very severe dysphagia. The ulceration was cleaned first with peroxide followed by an application of Acid Trichloracetic Pure. Application was made with a curved wooden applicator, no cotton being used. The patient said that there was no pain but a sense of heat which passed immediately. Within forty-eight hours the ulcer was seen to be clean and already filling with granulations. The pain was gone and she could swallow any kind of food or drink. She has since gained markedly in weight, and a cough which was present at that time, has stopped.

In all, eighty-six patients in the Los Angeles General Hospital, and twelve private patients, have been treated with results uniformly good. The ulcerations healed promptly and the patients were able to swallow in comfort.

The location of the ulceration does not influence the treatment in any way. The acid is easily applied and frequent applications are not necessary. No routine is followed as to a definite interval between treatments. They are given as indicated by the condition of the ulceration. As a rule, all that is required after the first application is a touching up of small areas that have not been as prompt in granulating as the rest or the ulcer was deeper. With subsequent applications these areas are stimulated and become even and smooth.

The applicator used is the ordinary seven-inch wooden applicator. No cotton is used. These are boiled in the sterilizer and while yet hot are moulded into the regular curve of a laryngeal applicator. For this purpose, we make the outline with long finishing nails, on a piece of inch board, using a double row, leaving room between the rows for the applicator. When they are dry they retain their curvature and may be used in the regular laryngeal mirror handle.

When complete coverage with granulations has taken place, it is a good practice to make regular applications twice a week of Chaulmoogra Oil 20% in Olive Oil in order to establish fibrosis and thus render the area firm and hard. As a rule this oil is used for two months.

The action of Acid Trichloracetic is escharotic. The tubercular ulceration is a broken down miliary tubercle, now filled with detritus which is very

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hard to penetrate. Pus and bacteria form a combination which perpetuates the ulcer and permits it to enlarge.

This mass is rapidly penetrated by the acid, instant coagulation takes place down to normal tissue, causing the mass to slough within a few hours, leaving a very clean even surface.

Following this process, granulation tissue begins to form within a very few hours, eventually covering the ulcer completely. Not more than four treatments have been given to any one ulcer in order to produce repair of the ulcerative area and eliminate the dysphagia.

There is no local inflammatory reaction following treatment and the action of the acid is strictly confined to the ulcer. As many as eleven miliary ulcers in the larynx have been treated at one time without distressing the patient. The very small ones disappear without forming granulation tissue.

In the treatment of Vincent's Angina with the ulceration of the tonsil, no more than two treatments have been found necessary to produce a clean and completely granulated field.

The following cases are mentioned as typical:

	Location of Ulcer	*Treatment Date*	*Result*
1.	Left Ventricle	8- 1-26	Very painful deglutition.
	Lateral surface	8-13-26	Pain gone. Ulcer clean.
	Left Arytenoid		Granulation started.
		9- 6-26	Healing complete.
2.	Transverse quarter	9-28-26	Extreme pain on swallowing.
	inch below tip of	9- 5-26	Very little pain. Ulcer much
	Epiglottis, poste-		cleaner. Granulating except
	rior surface. Much		right end of line.
	pus.	10- 1-26	Granulation slow right and
		10-15-26	Ulcer quite clean.
		10-21-26	No pain. Granulation complete.
3.	Ventricle near right	9-24-26	Painful deglutition.
	vocal process.		Ulcer very dirty.
		9-28-26	Granulating. Much less pus.
		10- 1-26	Granulation complete.
4.	Lateral surface	9-28-26	Painful deglutition. Much pus.
		10- 1-26	Ulceration cleaner.
			Beginning granulation.
		10-12-26	Granulation complete.
		10-26-26	Healing complete.
5.	Posterior surface near		Unable to swallow anything but
	tip of Epiglottis.		liquids.
	Considerable pus.	6-11-26	Eating comfortably. Ulcer clean
			and granulating.
		6-22-26	Eating well and gaining weight.
			Granulation complete.
		10-26-26	Granulation holding and fibrosis
			increasing.

The bibliography on this subject covering publications in the United States, describes a treatment using dilute solutions varying from 5% to 20%, covering a period of four to six weeks. Very few of the patients seen by us would have lived that long waiting for a cure.

While not claiming priority for the use of Acid Trichloracetic, I have not been able to find any mention in American publications regarding the use of the acid in full concentration on tuberculous lesions.

However, within the past week, and since this paper was written, I have received from the library of the Surgeon General, Washington, a copy of the French Review of Ear, Nose & Throat, Vol. 43, 1922, page 860, in which D. Bonain of Brest writes regarding the use of Acid Trichloracetic Pure in the treatment of Tuberculous ulcers of the Nose, Pharynx and Larynx.

He quotes Okouneff of Petrograd, who, writing in a French journal in

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1905, described his experience in its use dating back to 1895. He used it in treating erysipelas and any kind of ulceration, either on the skin or mucous membrane and is surprised that it is not used more generally as he says he has found it to be a specific in these cases. Bonain, giving his experience, is also surprised that it has not been used more in Europe and comments on the fact that its use has not spread outside of Europe at all.

He seems to have really started its use during the World War, using it in the treatment generally of tuberculous ulcers and sinuses and declares it to be a specific in these conditions. He goes into its chemistry very thoroughly and then describes his technic and results.

He uses a metal applicator wrapped with cotton, first anesthetizing the parts to be treated. This latter we have never found necessary. He declares that in many cases a single treatment has cured the ulcerations. Says the action is not deep and that in deep ulcerations he uses the actual cautery. This we have never found necessary but have rather given a few more treatments. Instead of the cotton wound applicator we have used the bent wooden applicator as described and feel that the dose can be better regulated in that way.

Discussion opened by Dr. Graham:

Trichloracetic acid is one of the most valuable cautery media for superficial cauterization that we have. The fact that it may be so easily localized in its action renders it a doubly valuable agent for the larynx, and Dr. Davies' original method of application renders it a practical every-day medicament. He is to be congratulated on his interesting paper.

Dr. Jos. Goldstein, Los Angeles:

Dr. Davies should be given credit for calling our attention to this valuable aid in the treatment of laryngeal tuberculosis. It has been my pleasure to see some of the results of cases under Dr. Davies' care in the Los Angeles General Hospital. It is indeed interesting to note how quickly patients are relieved of their pain and dysphagia. Such a drug, and the exact technique of its use, should be utilized more by those treating tubercular laryngitis. I should like to see it used more in the future.

Dr. R. M. Moose, San Bernardino, Calif.:

I would like to make a few remarks on this work of Dr. Davies because it appeals to me as being very important. I also had the opportunity of seeing some of the cases demonstrated by Dr. Davies and Dr. Wood at the Los Angeles General Hospital. The results were actually marvelous. Those patients previous to the treatment were unable to eat and were dying because of the inflammation and pain, but after the treatment with the Trichloracetic Acid were soon eating with comfort and gaining weight.

I wish Dr. Davies had taken more time to explain the local action of Trichloracetic Acid on the normal mucuous membrane. The action of the acid is quick and superficial and does not cause the dangerous Necrosis as we were taught to believe.

Dr. Davies (Conclusion):

Trichloracetic Acid on the skin is very painful. On the mucous membrane it is absolutely harmless and painless.

I have applied Trichloracetic Acid to my own lips and tongue to find out what it was all about and when touched to the mucous membrane it immediately coagulates. After first application the ulcer is clean of all pus. The pure acid is used, don't dilute it. It ruins it to add water and it is not necessary. There is nothing to be alarmed over at all and the patient will come back and tell you 24 hours after how much better he can swallow. You will get a lot of satisfaction from the use of pure Trichloracetic Acid in Tuberculous Ulcerations.

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FURTHER OBSERVATIONS ON THE ETHMOIDAL PROBLEM

ROSS HALL SKILLERN, M. D., Philadelphia

Some years ago, to be exact, in 1920, I presented my own experience and incidentally views on this subject, thinking at the time, if indeed I gave the matter any thought at all, that it would attract but passing attention and merely be classed as the personal views of an individual observer. Basing my opinion on the unsuccessful and in many instances harmful results following extensive surgical work on the ethmoid body proper, I advocated primarily the complete removal of the middle turbinate without interfering with any portion of the ethmoid capsule. This, of course, pertained only to those cases in which no surgery had been previously attempted or at least the middle turbinate and underlying cells were, even though diseased, to all intents and purposes intact. Unless this obtained, the very purposes of the intent were defeated. These purposes, of course, being two-fold; first, to install thorough aeration to the entire ethmoid, not only in the middle nasal passage below the insertion of the middle turbinate, but also around the superior turbinate and in the spheno-ethmoidal recess, through the removal of the underlying body which acted more or less as a barrier to that region, namely, the middle turbinate. Second, to permit the infected but intact ethmoid capsule to either rid itself of the infection through aeration and drainage or to react against the infecting organisms as to cause it to become localized in one or more cells or areas, thus making the infection amenable to logical and not haphazard surgical treatment.

Since that time further experiences have proven that there has been more in this assertion than I had hitherto dreamed. It is now a well-known fact, as many of our sad results will attest, that even under the most favorable circumstances the ethmoid capsule reacts badly to surgical interference and, indeed, directly as to the extent. In other words, the more intentionally the exenteration, the greater the liability of ultimate failure, and in the same manner, the less the infection or area of pathological changes, the greater the harm results in direct proportion to the extent of the surgical procedure. This statement may seem involved, but to use an old adage in a new light, one may say it is better to send a boy to do a man's work than a man to do a boy's, as far as the surgery of the ethmoid is concerned. In other words, it is better to do too little than too much. I appreciate that having made these statements, I am in the same position as the witness when the lawyer asked him if he still beat his wife. However, I do not propose to try to explain away the meaning of these remarks, but endeavor in the course of this presentation to show my reasons which if coinciding with your own experiences, they may also meet with the possibilities of your approval, if not actual endorsement.

Now in the first place, I am assuming that we have one of the common garden varieties of ethmoid cases to deal with where there is little to be seen on direct rhinoscopy outside of a juicy muco-purulent ethmoid region with some general hypertrophy but no signs of thick creamy pus streaming down in a given place which constantly reappears after wiping away as some of the text-books would have us believe. Only during attacks of so-called acute colds in the head (which these patients complain of with great frequency and which are, of course, only acute exacerbations of their ethmoidal infection) do we see any appreciable quantities of true pus such as is noted from the frontal or maxillary and even then is mixed with or accompanied by considerably larger quantities of a translucent mucoid secretion. Little or no headache or other extraneous symptoms are present and between the intervals of these acute flare-ups, the discharge from the nose, particularly that collecting in the naso-pharynx during the night and manifesting itself on arising in the morning, is the one great symptom that most annoys the patient and in all likelihood the one which has brought him to you.

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This, Gentlemen, represents the class of cases together with the preliminary surgery and after treatment that I wish to bring before you this evening for your consideration. We will assume that such a one has come to us and placed himself unreservedly under our care. We will also assume, mirabile dictu, that no surgery has been attempted, for in the very nature of the case it is a chronic one and it is indeed strange if he has not already passed through other hands. Straightaway on examination we of the old school would immediately think of that rhinological postulate, "Find the pus and follow it to its source." All right, try and do it. If one persists in his attempts to uncover the exact source he will soon find that to it the proverbial needle in the hay stack is as evident as a wart on the end of a nose. Pus, if any is seen at all, is either in the lower part of the nose or if above, is coming from everywhere in general and nowhere in particular. Even the naso-pharynx gives us but little information on this point. Suction, by means of negative pressure, will oftimes bring it out, but even then after it has appeared, the question arises, Where does it come from? Only very rarely and in the most exceptional cases with wide noses and a small middle turbinate could one judge with any degree of precision even the particular area from which this secretion was drawn, at least such has been my experience. Despite the fact that we have not been able to prove with any degree of accuracy from which cell or cells the inflammatory product has been secreted, nevertheless the patient under our manipulations has undergone more or less improvement and is quite sanguine of ultimately being relieved of his affliction. As time goes on, however, the true status of the condition becomes more and more apparent to both the physician and patient. The former sees that despite his treatments the appearance of the inter-nasal picture changes but little, while it gradually dawns upon the patient that at every little lapse in the continuity of the treatment, he seems to catch a fresh cold which leaves him in much the same condition as when he first came and the treatments were primarily instituted. Becoming impatient he insistently complains of the awful discharge and nasal stoppage which seems to grow in their intensity, treatments giving only temporary relief. This place has hitherto been the point where one actuated by the desire to relieve his patient in the shortest possible time, permitted his feelings to prejudice his better judgment and with the hope that fathered the conviction, flew to surgery, oftimes I am sorry to say, widespread and haphazard, the result of which all of us are or have been intimately familiar. As I say, once upon a time extensive surgery, that is, an attempted complete intra-nasal exenteration of the ethmoid, was indicated when this impasse between the ethmoidal infection and the attending surgeon prevailed.

Once upon a time, yes, once upon a time red flannel underwear was all the rage, flies were swarming in every dining-room and we only took a bath on Saturday night. But this has all changed, so it should be changed as far as the ethmoid is concerned; but I am getting ahead of my story. The Chinese have a maxim, Prudence will carry a man out of his difficulties, but the impetuous will find each step the more difficult. I do not know of any better piece of advice when we have reached this place in the therapeutic handling of our ethmoid case. We have already learned that by applying surgery we have only been cohabiting with chimeras and begetting specters, for who indeed has ever contemplated an ethmoid some months after a so-called complete exenteration with its conglomerate mass of hypertrophied and scarred mucosa, fibrous tissue and bone bathed in pus and carpeted with crusts that has not indeed seen a specter and a very horrid one at that, particularly if he himself had performed the operation?

It will then be seen that to resort to extensive surgery at this time with the hope of a radical cure is the acme of fatuity, but something must be done and very quickly at that if we hope to retain the confidence of our patient. Very well, let us consider the case from the point of view of what actually confronts us and what simple measures can be applied to correct or remedy the basic etiological factor, as the case may be. We have confronting us a

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unilateral infection of the ethmoid, the extent of which is undetermined, with intermittent exacerbations of discharge; sometimes a thin watery secretion, at intervals becoming thick and purulent, then reverting again to its original thin and acrid consistency, the middle turbinate being intact. Experience has already taught us that aeration and drainage are as essential to the ethmoid mucosa, in order to rid itself of infection, as oxygen is to the lungs and of the two aeration is far the more important. If we have good aeration the drainage will take care of itself. Well, what prevents the inspired air from freely circulating around, over and through the cells of this diseased and inflamed labyrinth. First, the body of the middle turbinate, and second, the swollen and oftimes polypoid mucosa. Very well, what are then the indications to bring around this desired aeration? Naturally to remove the obstruction thereto, namely the middle turbinate. Let me say here that this is the point where I disagree with some of my friends who consider this structure of great physiological moment in nasal respiration and who make every effort to preserve it intact by performing an excavation of the cells between it and the orbital plate. I cannot agree with them in this respect nor can I subscribe to the importance of all the functions which they attribute to it. It seems to me if one accepts that aeration is the prima disideratum in the ultimate cure, that to keep the middle turbinate in place defeats the very object of the intent, for this body acts as a barrier in directing the inspired air with its high oxygen content from and away from the posterior ethmoidal regions. The middle turbinate is then removed in its entirety by the method most favored by the individual operator. Personally I prefer the scissors and snare removing as much as possible with the first constriction of the wire and leaving permanently the posterior end to about the size of a small pea. I have found by leaving this portion that hemorrhage both immediate and post-operative has become a thing of the past. This is due to the fact that the large naso-palatine artery branches just behind this point so that by avoiding the extreme posterior end of the turbinate, injury to this artery is also avoided. The removal is made on a line with its insertion into the ethmoid body proper, thus keeping well below the terminal fibrils of the olfactory nerves and avoiding the possibilities of the dangers incurred in opening their peri-neural sheaths which, of course, lead directly into the sub-arachnoid space.

As soon as this turbinate is removed, our operative technique should be suspended except perhaps for the removal of ragged portions of bone or mucosa which happen to remain. The main point is to leave the body of the ethmoid absolutely intact as much as it pains us, for who has not seen the polypoid changes and even small polypi revealed around the neighborhood of the bulla which seem to so hospitably invite their removal as soon as the overlapping turbinate is out of the way. Our enthusiasm in this regard may be the very cause of our undoing as I have found that to continue at this stage is to distinctly overdo it for in endeavoring to remove this redundancy of hyperplastic tissue we not only take it, but also a certain amount of healthy tissue in the immediate vicinity, both of which would have in all likelihood returned to normal following the aeration made possible through the removal of the turbinate. So that our one endeavor is to completely and cleanly resect this structure and discontinue any further surgical manipulations, at least for the time being.

Now just as in a gastro-enterostomy for duodenal ulcer, here also this operation should be considered as a preliminary procedure to subsequent treatment, for without one the other loses most of its virtue. In four or five days or as soon as healing has well set in, the local treatments should be instituted under the following hypothesis. First, that the extent of the infection through the ethmoid body is unknown and second, though marked hyperplasias be present, even to actual polyp formation, the area of subsequent tissue resolution to normal through the newly created aeration and under appropriate forms of treatment is also unknown. Our first endeavors should be directed towards ascertaining, as far as humanly possible, the extent of the infection through the ethmoid labyrinth, after which treatment can be applied

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that may be termed fairly scientific and does not favor the haphazard methods which consisted of all that we have hitherto had under our control.

About five days, then, after the removal of the turbinate, on inspection, the whole ethmoid capsule on that side has undergone a marked change. Instead of a mass of acutely infected, swollen and hypertrophied tissue, the entire picture has changed, giving one the impression that the inflammation has not only markedly lessened, but also shows a tendency to congregate in certain places with areas of mucosa intervening which apparently have entirely regenerated, especially if marked hyperplasia had been uncovered when the turbinate was first removed. Even small solitary polypi have reduced themselves almost to disappearances. Now is the proper moment to use our method for the purpose of ascertaining the spread of the infection. This can be done by applying some substance which will not only tend to draw the inflammatory products out of the infected tissues, but also to act as a germicide as well as have a beneficial curative action on the diseased mucosa. This I appreciate is a large order but I feel convinced we have found true panacea in tampons about the size of one's thumb saturated with a strong solution of colloidal silver. Many firms manufacture such a drug which goes under the name of argyrol, silvol, carcentos, silloid, etc.; all are claimed to be non-irritating so that I shall play no favorite. There are, however, several conditions in their application which must be observed. First. The solution must be strong (25-50%). 2nd. The solution must be fresh. 3rd. The solution should be hot or at least warm. 4th. The tampons must remain in place from four to six hours. These tampons while in place act in a similar manner to a hot poultice, but in a twofold manner. Firstly, they tend to draw the inflammation toward the surface and secondly have a decided bactericidal action on the infecting muco-organism as well as possessing a healing quality beneficial to mucous surfaces.

Briefly, then, the entire method is carried out as follows: Both nares are thoroughly cleansed with warm, normal saline solution. A suitable piece of cotton is fashioned into a tampon about the size of one's thumb and partially soaked in a fresh, warm, strong solution of the colloid silver, then gently pressed into the upper portion of the nasal chamber until it covers the entire ethmoid region. The tampon, as I said, is permitted to remain in place from four to six hours. These treatments are carried out every other day until the inflammatory symptoms have greatly subsided, then gradually tapering off until they cease entirely. The length of time varies with the individual case, but roughly speaking one can count on from four to six weeks for the average case.

These cases that we have just described, it must be remembered, were those in which no surgery had hitherto been attempted. Now let us consider that group in which multiple operations had been performed and the whole ethmoidal region represented a thoroughly disorganized and semi-dense suppurating mass with all landmarks as far as the ethmoid itself is concerned, more or less obliterated. Further surgery merely adds fuel to the fire, as the resulting granulation tissue soon becomes reinfected, making the condition worse with each succeeding operation. If such a case comes under our care we must take it up from a systematic point of view and first rule out suppuration from the true sinuses. In the presumption that this has been accomplished we now endeavor to ascertain from which portion of the ethmoid the inflammatory exudate is secreted. Sometimes this is made possible by using the tampons and on removing them noting which portion is covered with pus or greatly decolorized. If the diseased portions can be brought to light by this procedure, appropriate treatments to those localities can be immediately instituted. Suppose on the other hand the precise source of the purulent discharge remained undisclosed, the pus appearing to be everywhere yet coming from nowhere. These cases require the most thorough and far-reaching surgery that we are ever required to practice intra-nasally with reference to the ethmoid. I feel and have felt for some time that to reach all the cells that are accessible to intra-nasal manipulations, it is essential to re-

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move the uncinate process from its insertion to its end. I am also quite aware that with even this structure out of the way the entire ethmoid labyrinth cannot be removed endo-nasally, but as a matter of fact this is almost never indicated as all the cells are not always affected, at least to the same extent as to require complete exenteration. In any event, after the uncinate is out of the way, from one quarter to three-eighths inch more room is gained which may give us just the amount of space necessary to successfully carry out our applications. The hot tampons are continued here just as before although the occasional removal of polypoid or excessive granulation tissue may from time to time be found necessary. Improvement, however, will surely come and if the final results do not measure up to one hundred per cent, nevertheless, if we have relieved the patient to such an extent that he says, "Why, Doctor, the little inconvenience that I now have is nothing to what it was before the treatments," we should be content with our efforts, for no one can expect an ethmoid which has been torn to tatters by infection and instrumentation to ever functionate as a normal one and not be a place of minor resistance for every cold to which the patient is subjected.

And in conclusion, Gentlemen, let me state that I firmly believe as time goes on we will no more look upon our treatments in the light of a hope rather than a faith, but will finally agree upon a standard method of handling these cases with such uniformly happy results that the subject will no longer be termed "The Ethmoidal Problem."

Discussion opened by Dr. Harris P. Mosher, Boston:

Mr. Chairman and Gentlemen: This is a paper which gives us food for thought. It is a scintillating paper filled with skill and the writer began by saying "we of the old school." I am of the same school. I had down: it is a gray-haired paper, and a gray-haired paper is and should be based on abundant experience. I did not have the opportunity of reading this paper; he kept it away from me as he wanted my reaction. I will relieve him of this point by saying that I have never before so thoroughly agreed with him as I do at the present time. The class of case which he describes is the transient more or less beginning case. He gave the body of the paper up to treatment of that type then switched over to another type.

Operation in the Ethmoid with slight indication always has been a problem to me. These are beginning slight cases, but Gentlemen, you can make them continuous cases; by operative interference you can make them permanent cases. One of my assistants said some years ago, I envy him many of his sayings, "Once a sinus case, always a sinus case," and that applies to a case that has been attacked early where the symptoms were slight. I have never seen a cured case where the operative technique has been worth while for the patient. Sometimes it has been worth while financially for me but the case is never entirely cured and we should not expect to entirely cure them.

That aeration is most important by removal of middle turbinate to get, goes without saying. And having that and knowing that, I do not see why we should not rely on aeration to work its more or less miraculous effects on the ethmoid. I am very much obliged to Dr. Skillern for giving us a rational method of treating these drainage cases, these cases which from early surgery can become permanent cases. I have used argyrol, sylvol, and I cannot say that I have the confidence in it that Dr. Skillern has, but it fits in very nicely with his scheme. I think he is over enthusiastic about it, however. Ethmoid surgery is in my hands a very disappointing surgery. There is a very large amount of truth in what Dr. Skillern says and I cannot for the life of me make out why anybody on slight indications wants to immediately attack the ethmoid surgically knowing he will probably infect it.

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THE RECONSTRUCTION OF A COMPLETELY DESTROYED AURICLE

HARRINGTON B. GRAHAM, M.D., San Francisco, Calif.

The opportunity of reconstructing the auricle occurs so infrequently that the operator seldom has a chance to learn how to improve his technic from personal experience. He must look to the description of the operation by others and so far the errors one may fall into have been very casually dealt with. I am attempting to describe two cases, one of my own, and the other by Dr. George Pierce of San Francisco, quite differently done, both of which could have been improved on by experience.

The important factors involved in the operation are:

1st—The tissue used must be in appearance and consistency similar to the original ear.

2nd—The support must be equal to the demands of keeping the new tissue erect and away from the skull.

3rd—The new ear should be as thin as is possible to make it.

If the tissues behind the ear have been uninjured, they are available for the reconstruction and even the hairy parts may be used, as they may be folded back in such a way as to bring the hair on the posterior surface, where it may be easily kept short. If this tissue is not available, the whole ear must be reconstructed from the skin of the chest, abdoman, or arm, none of which has the appearance of the original ear and can in no wise be as satisfactory as the scalp tissue.

The support may be celluloid, in which case there is a grave chance that it may later on be extruded. Of course, its thinness and malleable quality is a great asset. Personally, I prefer cartilage and have found the nasal septal cartilage satisfactory. The assertion that this is absorbed more frequently than is rib cartilage, has not been found to be so in my nasal plastic work; either may be absorbed, the determining factor being unknown to me. Gillies has suggested using rib cartilage in order to be able to excavate it so as to provide a support the shape of the former ear, but this does not appeal to me, as it is a difficult task and the shaping might increase the tendency to absorption. It is hard to get a piece of rib cartilage as thin as septal cartilage, and this is a very important factor, as far as the ultimate appearance of the ear is concerned.

The thinness of the new tissue will depend to a great extent upon the position subcutaneously of the transplant. If it is buried deeply in the tissues, the result must be a thick ear. A superficial position is a difficult one to obtain, but will pay the operator for his pains. Any tissue taken from the arm or chest must be thick, looking like a piece of putty when transplanted. However, time modifies its appearance so that under certain circumstances it may be necessary to use it.

My case was injured in a railway accident, the patient losing all but a portion of the lobule of the left ear. The tissues on the scalp were undamaged so that I could use the non-hairy parts for the reconstructed portion. My first step was to transplant a piece of septal cartilage the shape of the sole of a shoe to the subcuticular tissues posterior to the defect. I placed this directly behind the defect instead of above and behind, so that when I brought my tissues forward I did not have enough support to keep them high enough. Any cartilage either from the nose or rib, used to keep the soft tissues up and away from the head, should lie, by preference, in the upper one-third of the completed ear. It is difficult to get enough nasal septal cartilage to fill the demand, so that the lower portion of the lobe may be disregarded.

After leaving the cartilage in place for a month, I made a circular incision to the hair line, starting at the upper anterior part of the old ear. This incision was carried to the periosteum and the skin thus outlined, including the cartilage, was raised and folded in such a way as to have the

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upper border of the ear covered by a continuous layer of skin. The anterior portion was now sewed down to the tragus, and the defect on the scalp remedied by the customary undermining, reduction in size of opening, and a skin graft taken from the leg, which was brought well up onto the back of the new flap. Were I to do another, I should not fold the upper edge back, as in this case, but would use it entire, placing a graft over the whole posterior surface. This flap, if the cartilage were properly placed, would be quite sufficient to form a new ear without any further addition.

After another month, the lower part was incised, carried forward and sewed to the freshened lobule, another graft being placed posteriorly. A few minor operations were done to smooth things out, and an attempt made to stiffen the upper border somewhat by the introduction of celluloid, but this should not have been necessary.

Dr. Pierce proceeded quite differently with the reconstruction in his case. He transplanted the rib cartilage placing it in position subcutaneously through a small incision above the ear. After six weeks a complete incision was made outlining the new antihelix which was then raised from the periosteum and a skin graft placed so as to cover the whole denuded surface, with stent in the center. The edge was then sewn back into its place of origin and again freed after ten days. He then brought a tube pedicle flap from the chest up to back of the ear and draped this over the new edge, forming a new helix, which is quite effective. This procedure enlarges the ear so that it is fully as large as the other, and when completed will be nearly the same shape.

The ear that I constructed is considerably smaller than the normal ear, due to the fact that I did not carry my incision high enough on the head, that I folded too much tissue back in order to get a smooth border, and that my cartilage was not placed high enough. All of these errors should be avoided in future work. The assertion of one author that no reconstructed ear is worth while, is certainly a false statement, and the time is coming when we will be able to replace a destroyed ear by one which is nearly the equal of the original. Concentrate this work in the hands of a few surgeons and the technic will soon be developed satisfactorily.

Discussion opened by Dr. Harter, Seattle:

Mr. President and gentlemen: I am sure that you men, as I, appreciated hearing Dr. Graham with his wide experience tell us about the building of this ear. This is one of the most difficult problems encountered in plastic surgery.

The difficulties of rebuilding an ear vary as to how much of the ear is present and whether it is a congenital deformity or the result of an injury. I had a case of a boy, 17 years old, who accidently shot away most of his left ear. The upper portion of the helix and the lobule were left and I rebuilt this with a sliding flap from the scalp. One month later I inserted a thin strip of costal cartilage and patient has a fairly good ear.

Recently I saw a man who was badly burned when a child. He appears to have an ear canal, but no auricle, but on close examination the auricular cartilage is present beneath the skin. This case could, I believe, be fairly easily repaired. I would take just enough skin to make the turn over the auricle and fill in the raw area with pedicled graft from the arm. A graft from elsewhere, even Thiersch grafts, could be used.

Dr. Graham's use of septal cartilage for support of the ear was very encouraging. I have always heard that septal cartilage being fibro-elastic cartilage was quickly absorbed. This gives us one more source of support for soft tissues.

Dr. Mellinger, Santa Barbara, Cal.:

I have never reconstructed an ear, but would like to report a case.

In a boy one year of age, the folding of the skin which separates the ear from the scalp had failed to take place. The auricle could be palpated be-

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neath the skin. Except for the lobe, there was no ear visable. The correction was made by a semilunar incision back of the ear far enough to allow the suture line to come in the bottom of post auricular fold. The scalp was moved forward enough to meet the line of suture. The web over the auricle was carefully dissected away and a perfect ear was the result.

I think Dr. Harter might use this method in his case.

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HYPOPHYSEAL TUMORS IN RELATION TO THE EYE AND NOSE

JAS. M. PATTON, M.D., Omaha

The importance of ophthalmologists and rhinologists considering disturbances of hypophyseal origin is emphasized by the findings of neurologists, neurological surgeons, and radiologists, as well as of men in our own specialty. Dr. Charles Frazier[1] found failing vision was the first and predominant symptom in 71 per cent of his 177 cases, with 27 per cent complaining principally of headache. Visual disturbances, according to Cushing[2], are the most common, and needless to say, the most serious of all neighborhood signs of hypophyseal disturbances, while troublesome epistaxis is very common and the bleeding may be excessive. Keegan[3] states that the problems associated with the pituitary gland concern the ophthalmologist in the visual field defect and the rhinologist in the fossa enlargement which requires pituitary decompression through the nose. The headache, which may be present without visual field defect, or marked pituitary enlargement, also calls for careful consideration before sinus operations are undertaken for their relief. J. Herbert Fisher[4] places visual phenomena as among the most prominent symptoms of pituitary disease, while G. de Schweinitz[5] considered this relation to be of sufficient importance to select it as the subject of the 1923 Bowman Lecture.

Consequently, if 71 per cent of the cases are first brought to the attention of the profession on account of defective vision, and 27 per cent of them have headaches which may be interpreted by the laity as due to sinus disease, etc., it is certainly worth our while to consider clinically, at least, this structure which lies within our field, whose pathology affects the vision and upper air passages, and for the correction of which, whether it be medical or surgical, the sufferer logically looks to us for relief.

ANATOMY

Beyond recalling a few outstanding facts with which we should be familiar, a discussion of the anatomy is not necessary. Dr. de Schweinitz[5] has given a most thorough presentation of this phase of the subject. We should remember the origin of the anterior and posterior lobes. The former is from the pharynx, the so-called pouch of Rathke. The latter is from the floor of the third ventricle, to which it is connected by the infundibulum, or pituitary stalk, and histologically is brain tissue. The two lobes of the hypophysis are ductless glands, subject to the disturbances and influence of other ductless glands. Both lobes are lodged in the bony, cup-shaped sella turcica, at the base of the brain nearly four inches from the surface, by any direction of approach. The sella is lined and also covered by a layer of dura. The covering or diaphragm fenestrated for the passage of the infundibulum varies from a narrow band of rather delicate membrane, only partly covering the sella, to a firm tissue roofing over the space with only sufficient opening for the passage of the infundibular stalk. The sella varies considerably in size within normal limits. Pottes [6], checking up the measurements of 41 normal heads, of which radiograms had been taken for other reasons than hypophyseal diagnosis, found it to vary from eight to sixteen millimeters anterior-posterior, with an average of 10.8; and from five mm. to 10½ mm. in depth, with an average of 7.7. An examination of 29 skulls failed to show any definite relation between the measurements of the skull and the size of the sella turcica. None of these cases showed any symptoms referable to hypophyseal disturbance.

The course of the optic nerves varies in location with reference to the hypophysis. As shown by de Schweinitz[5], the intra-cranial portion of the optic nerves may be long and the commissure directly above (79%), or even back of the hypophysis (4%), or this portion may be short (5%), with the anterior portion of the commissure resting on the chiasmatic sulcus.

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("In 12% the entire chiasm rested over the diaphragm and sub-ajacent hypophysis.") Furthermore, the basal surface of the chiasm may be in contact with the dorsal surface of the diaphragm or there may be as much as 10 millimeters of separation. The position of the internal carotid, anterior cerebral, and anterior communicating arteries vary with reference to the commissure and hypophysis and with the previously mentioned anatomical relations may be a factor in the production of the varied symptoms we are accustomed to attribute to hypophyseal disturbances.

PATHOLOGY

Of the pathologic changes perhaps the simple hyperplasias are most frequently overlooked. As we well know, the ductless glands are subject to temporary enlargement as in puberty, pregnancy, and other physiologic disturbances. In some, for example, the thyroid, the temporary increase in size is harmless; but in the hypophysis due to its intimate relations, a very slight change in size may easily produce disturbances of vision, headache, and changes in the visual field which are distinctly characteristic.

Lancaster and Carvill[7] examined the fields of 61 pregnant women (otherwise normal) within a month of parturition and found bi-temporal contraction of the visual fields of a greater or less extent in 89%. They refer to the report of Erdheim and Stumme who found from an examination of 150 subjects the average weight of the hypophysis in the non-pregnant woman to be 61.8 grains as against 84.7 grains in the primpara and 106 in the multipara, indicating rather positively that there is a normal enlargement of the pituitary in the pregnant woman. Finley[8], after an examination of 31 cases with narrowing of the temporal fields in 22, attributes this change to pressure exerted by the normal hyperplasia occurring during pregnancy. In two of our cases (12 and 22) the early evidence of trouble was first noticed during pregnancy, while case 7 suggests menstrual irritation as the primary cause of a possible pituitary headache. As mentioned by Potts[6], enlargement of the hypophysis should not be classed with other intra-cranial tumors, for we not only have to consider the effect of pressure on the neighborhood structures, but must in addition recognize general changes due to the deranged activities of the gland.

There are various classifications of the tumors of this region. As to location, they may be extradural, within the sella and walled off by a firm diaphragm. They may be primarily supersellar, pressing upon or invading the brain substance, or a tumor primarily intra-sellar may extend through the diaphragm until the greater part of the mass is within the dural envelope. As to type, different observers place the solid tumors (adenomas) at about 80%, cystic degeneration at 20%, the latter most frequent in the supersellar group. Although the adenomas originate from the anterior lobe, Frazier[1] finds in many of his cases evidence of posterior lobe disfunction; for example, amenorrhea, accession of fat, somnolence, and the like. The adenomas are essentially benign and even though malignancy may develop, it is usually not of the rapidly destructive type. Carcinoma, glioma, and syphilitic gummata may involve either or both lobes, but they are the exception. As the tumor enlarges it usually presses upward and forward, the direction being determined by the character of the diaphragm and the position of the chiasm. Even after a sub-sellar decompression, the upward extension of the tumor may continue.

DIAGNOSIS

The diagnosis is based upon a rather large group of physical findings and symptoms, some rather varying and indefinite as headache. Others, e.g., skeletal changes, acromegaly, gigantism, or fat dystorphy, are more or less constant and characteristic.

For convenience, we recognized three groups of symptoms. First, local. The X-ray may show the sella to be very small, with a narrow roof. This, with a charactertistic headache, should arouse suspicion. The sella may be

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relatively large. This alone, as stated above (Potts) may have little significance, but when associated with evidence of hypophyseal disturbance, especially if the clinoids are eroded or absent, it is not only a diagnostic sign of value, but may indicate an active, or possibly a malignant type of tumor.

Timme[9] describes pituitary headache as within the head, prostrating, boring, or bursting in character, with a subjective sensation of a moving mass within the head. The patient describes the pain as between the temples, below the crown, back of the eyes, or between the bridge of the nose and the occiput, all lines converging to the sella. He further states that the headache comes on after fatigue, fasting, in connection with the menstrual periods, after meals rich in carbohydrates, mental anxiety or other debilitating conditions. Williams[10] thinks that every case of chronic headache should be studied with the thought of hypophyseal etiology, and not dismissed as trifling or psychological. If the 27 per cent of cases who consulted Frazier complaining of headaches as an initial symptom (75%), according to de Schweinitz[5] who found it a conspicuous symptom in a hundred and twenty cases of pituitary disturbance determined by operation or autopsy (Bowman Lecture) is a fair average of cases in general, it certainly behooves us to consider it most carefully. Symptoms arising from changes in the upper air passages are of value. Cushing[2] has noted an occasional unexpected discharge of mucous into the pharynx in cases of pituitary disturbance, and reported a case of true cerebro-spinal rhinorrhea which must be distinguished from the rhinorrhea of sinus origin. Changes in the sub-sellar wall of the sphenoid, due to pressure or erosion, may result in a partial or complete obliteration of the sinus cavity, with the tumor visible in the retro-nasal space. He warns against treating some cases of optic atrophy for supposed sinus disease. In any case of suspected pituitary involvement, a careful retro-pharyngeal examination should be made.

Although the above findings are a valuable aid in diagnosis, the symptoms incident to changes in the vision and visual fields are of the greatest importance. Due to the intimate relation of the hypophysis to the commissure, optic nerve, and in some cases the oculo motor nerve it is not surprising that at least 71% (Frazier) of persons suffering from the disease of the pituitary mention ocular disturbances as the first recognized evidence of trouble. The mechanism of reduced vision, scotomata, and field changes has been carefully presented by de Schweinitz[5], Fisher[4], Cushing[2], and many others; and we need only to recall that pressure from an enlarging hypophysis, the infundibular stalk and adjacent arteries, exerted on the optic nerve, tract, and chiasm, logically produces the characteristic or atypical ocular findings with which we are familiar.

From the usual direction of the line of force, the defects in the upper outer visual field are first to be noticed, followed as a rule by partial or complete loss of the temporal fields; but the pressure varies widely in different individuals, or may vary from time to time in the same patient as emphasized by de Schweinitz[5]. Homonymous bi-temporal hemianopsia is by no means the signature of hypophyseal enlargement (de Schweinitz[5]) and its absence should not divert our attention from the pituitary region. He emphasizes the instability of visual field boundaries and states that a concentric contraction present at one time may soon after be replaced by the so-called typical hemianopsia due to a shifting of pressure. Islands of vision may develop in the blind area of the field (Cases 2 and 9), scotomas may expand and contract. Various field alterations due to changes of pressure may readily lead the observer astray, not so much in diagnosis as in prognosis, and cause one to attribute improvement to a remedial agent which was entirely without influence. McCulloch[11] emphasizes the importance of outlining the field with very small test objects.

A blind eye, especially if the nerve is atrophic, with a quadrant field defect or hemianopsia in the other, should suggest hypophyseal disturbance; as should double optic atrophy with an enlarged sella even though there is not sufficient vision for field measurement.

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Scotomata, continuous with the physiological blind spot (Lilly[12]) are not characteristic, but are present in a certain number of cases, as are disturbances of motility as shown by diplopia and ptosis, and represent variations in the direction of pressure.

Of intra-ocular changes, atrophy of the optic nerve is most frequent. The appearance of the disc according to de Schweinitz[5] differs from that of simple atrophy from other causes. The comparatively even distribution of the pallor of the nerve head, and its somewhat waxy aspect with the suggestion of a yellowish tint, are well recognized characteristics.

GENERAL EVIDENCE

General signs and symptoms secondary to disfunction of the hypophysis are well known and need only to be mentioned. Hypo-function of the anterior lobe, if it occurs before puberty, interfers with the normal development of the individual, resulting in dwarfism, undeveloped secondary sex characteristics (infantilism), and in some instances an inhibition of the mental development of the child. If it occurs after puberty, there is a loss of sex characteristics in both the male and female. There is an increase in fat production and a tendency in the male to become feminine in appearance. Hyper-function of the anterior lobe in the young results in a great over-growth of the long bones, resulting in gigantism. In older individuals, it produces acromegaly. Dysfunction of the posterior lobe is thought by some to be a causative factor in menstrual disturbances, somnolence, diabetes insipidus, etc. We know that the extract of the posterior lobe of animals acts especially on smooth muscles, consequently blood vessels, uterine and intestinal structures, are stimulated by it. The physical evidences of dysfunction of the hypophysis vary widely in degree and expression, but any unusual skeletal changes should be investigated with this structure in mind.

TREATMENT

The treatment of hypophyseal disturbances may be considered under three heads. Namely, glandular feeding, radiation therapy, and operative interference. Practically all observers recognize some value in all three methods, but vary greatly in opinion as to their relative value. De Schweinitz[5] is convinced that evidence of the value of glandular feeding is abundant; but states that Frazier and Cushing are disappointed in the results of this form of treatment and Adson[13] has not seen as favorable results from glandular feeding as he would like. Pardee[14] has found that, even with an enlarged sella and eroded clinoids, endocrine treatment relieves the symptoms and from the literature he finds a slow though definite swing in the attitude of the profession toward glandular therapy. Hill[15] and Kern[16] report favorable results from glandular treatment. Timme[9], writing in 1926, administers the whole gland substance, varying from one-half grain to three grains a day. He gave injections of pituitrin for two or three days a week, and later one-half to one grain of thytoid once a day. He states further that in early cases, where headache and glandular symptoms are mainly emphasized, where vision is not greatly interferred with or the visual field much encroached upon and where the sellar deformity is not extreme, radiation therapy should be given a thorough trial before resorting to surgical measures, while Holloway[17] is of the opinion that surgical intervention should be the initial treatment, followed by radiation and gland therapy.

A case reported by Tyson[18] was first given a course of glandular feeding. A subsellar decompression was then done, followed by radium. The outcome was favorable, the patient having normal vision in one eye. This would seem like a logical procedure in cases requiring operative care. Tousey[19] in discussing Tyson's case, emphasizes the great advantage of an operation which permitted the radium to be placed in contact with the tumor. Pardee[14], Keegan[3] and Grant[20] remind us that in severe cases, especially acromegalics and in some supersellar cysts, that the vision may be reduced following radiation treatment. This was observed in two of our cases (8 and 23)

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followed later by improvement. X-ray, radium, or both have proven helpful in the experience of Braunstein (21), Fejer (22), Di Marzio (23), Dillarz (24) and others. Towne (25) has found that pharyngeal pouch tumors primarily super-sellar and usually cystic, are not favorable for X-ray treatment, while many anterior lobe adenomas respond favorably. All adenomata should be given this form of treatment and operation indicated only when the vision and fields fail to respond. Frazier (26), Daily (27) and Adson (13) advise radiation treatment, especially radium as a post-operative measure. Davenport (28) reports two cases; one greatly improved after one massive radiation treatment, the other similarly improved following fractional doses distributed over several months. The first was improving while under observation before radiation was instituted. He thinks the improvement in this case may have been due in part to a spontaneous shrinking or decompression of the tumor. De Schweinitz (29) felt there was distinct value in radiation treatment, but it should be carefully administered, and that occasionally very satisfactory results were obtained. He found its greatest field of usefulness was in association with operative procedures.

J. Herbert Fisher (4) writing in 1911 said: "I am not competent to discuss operative procedures. Difficulties are enormous and the results deplorable;" and again in 1925 (Fisher) (30): "One should utter a word of warning to those who think that operative interference to the pituitary body is to be lightly undertaken by the neurological surgeon." He mentioned a number of cases in which great advantage had resulted from the administration of glandular therapy. His plea was, that operative treatment should not be employed too hastily, and its use limited to those with very special experience and the special gift to use the experience. Sargent and Carmichael (31), writing on the treatment of pituitary tumors, after some unsatisfactory experience, abandoned the trans-sphenoidal route in favor of the trans-frontal operation. In their experience the latter is almost free from danger, and they reported 17 cases operated within the last few months without a death. They stated that the earlier one can operate and the smaller the tumor, the safer the operation. Where the consistency of the tumor is soft it is removed with a suction apparatus, a glass tube slightly curved being introduced through an incision in the tumor capsule. The other end is connected with an aspirating bottle. When the growth is of a harder consistency, it is broken up with a currette, and the fragments sucked out of the capsule. They felt that operation was justifiable, not only in cases where there was progressive loss of vision, but also where patients were suffering from headache of great severity, which could not be relieved by any other means than incising the capsule of the tumor. Gordon Holmes (32), while recognizing the fact that fields of vision or visual acuity may improve spontaneously, or as the result of administration of thyroid or pituitary extract, or after radiological treatment, was of the opinion that operation was often necessary, when only slight changes of vision were combined with much suffering on the part of the patient, or marked mental or physical deterioration.

Timme (9) analyzed twenty cases in which glandular treatment proved effective, but stated that other cases with similar symptoms were not improved. His discontinued glandular treatment on the appearance of premonitary headaches, resuming the treatment when it disappeared. Pardee (14) finds that other endocrine glands may become over active in an attempt to take over the pituitary function. In cases of hypophyseal disturbance, when compensation is established the progress of enlargement ceases. He favors glandular therapy and feels that patients should have this benefit before undertaking operative treatment, to relieve the symptoms arising both from the pituitary and other endocrine glands. Calhoun (33) and Key (34) have found that glandular therapy is much more effective when combined with anti-syphilitic treatment, where clinical or serological evidence reveals lues; while de Schweinitz (35) states that in cases of glandular insufficiency, the efficacy of organo-therapy is probably inhanced by simultaneous admin-

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istration of mercury, preferably by inunctions, even though the presence of syphilis is not demonstrated by the usual methods. Furthermore, it is probable that a combination of thyroid and pituitary gland extract is more efficient than either of the extracts alone, and this combination, accompanied by mercury, is more effective than the extract of one gland, even though given with mercurial inunctions.

With but few exceptions, it is generally conceded that the sub-sellar operation is decidedly safer than the trans-frontal approach, but is of no value in relieving pathology that is primarily super-sellar, except in so far as the decompression may relieve pressure and increase the efficacy of radiation therapy. It is exceedingly difficult and often impossible to remove all of the tumor or cystic tissue by either route, rendering post-operative raying all the more necessary.

If a reasonable course of glandular and radiation treatment fails to check the progress and relieve the symptoms of a hypophyseal disturbance, operation should be advised before definite optic atrophy has developed. The earlier operative measures are instituted where conservative measurs have failed, the better will be the result.

The following (with the exception of Case 5) are cases observed by my colleagues, Drs. Harold and Sanford Gifford, W. F. Callfas, John B. Potts, and myself within the last ten years. Several cases of fat dystrophy (Distrophia adiposa genitalis) are not included. As we are considering this from the viewpoint of the ophthalmologist and rhinologist, I have grouped the cases according to therapy, indicated or employed, rather than to symptoms and probable pathology.

GROUP I

Case 1, 9261—Mr. C. B. E., age 34. Consulted us December, 1918, on account of failing vision. Right eye equaled perception of shadows, left eye 20/200 not improved by glasses. General history negative. He gave a history of the vision in the right eye failing rapidly during the first two weeks of the previous July. Sight of left eye had failed in last six weeks. There was decided physiological excavation of the optic disc, otherwise fundus normal each eye. X-ray showed large shadow around the sella which was obliterated and involved a large part of the center of the brain. Posterior clinoid process, sella and posterior part of the roof of the sphenoid absent. His family physician was advised to give him thyroid extract. He took five grains a day for one year, then irregularly for a couple of years. Was advised to continue taking five grains twice a day for two weeks out of every month. At our last examination, May 14, 1921, the vision was 20/20 in each eye with fields as shown. Improvement was evidently secondary to glandular feeding. No report since 1921. Field could not be taken at first examination satisfactorily, due to poor vision.

Case 2, 23358—Miss I. R., age 21, September 7, 1922. Had noticed poor vision for four or five months. Had small pox and operation for appendicitis during that period. Also operation for peri-nephritic abscess during previous year. Otherwise general history negative. Vision in the right eye was 20/200, left eye counting fingers. Had occasional temporal headaches, sometimes on top of head, but no superficial tenderness. Ophthalmoscope showed a decided paling of the outer quadrant of the left disc. There was bi-temporal hemianopsia with reduction of the left nasal field, but with an island of vision in the lower temporal quadrant of each eye. A roentgenogram showed the sella very much flattened with the posterior clinoids largely missing. No definite indication of enlargement of the sella or of intra-sellar tumor. She was put on intensive glandular feeding, which resulted after three months in a decided improvement of her visual fields, but without much change in the vision, although at our last examination, March 24, 1927, her vision with correction was 20/200 in the right eye, 20/70—1 in the left eye, with a nearly normal field in the right eye, irregularly hemianopic in the left. Both eyes show externally a fine rotary nystag-

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mus, while the motion of the optic disc is strictly vertical, except at intervals, when there is a distinct rotary movement. Discs are rather light, otherwise normal. Has dull ache in eyes at times, otherwise feels perfectly normal. No gain or loss in weight. Glandular feeding continued. From the history, it would seem that this patient after an initial improvement in vision and fields was relieved of headaches, with no increase of her symptoms over a period of five years. Of course, we cannot say positively that the glandular feeding was responsible for this, but from the fact that she was rapidly getting worse previous to her first examination and improved following the glandular feeding, it makes it at least worthy of consideration.

Case 3, 61625—Mrs. P. A., age 55. Consulted us April 11, 1927, with the history that her glasses were not satisfactory, unable to read comfortably. Left eye failed considerably in the last month. Vision in the right eye with correction was 20/40, left eye uncorrected 1/200. Ophthalmoscope showed both nerves pale, about equally. Fields, right incomplete temporal hemianopsia. Left complete temporal hemianopsia with preservation of fixation. X-ray showed sella turcia twelve to fifteen millimeters in extent. Clinoid processes erect, probably due to enlargement of pituitary gland. Referred to her local physician and oculist, who was advised to use glandular feeding, providing the condition was getting no worse, and pituitary extract hypodermically if administration by mouth did not seem sufficient. I am informed by her local oculist that she has not improved on the gland therapy and is considering operative interference.

Case. 4. 32751. Mrs. K. S. O., age 72. Consulted us September 4, 1923, complaining of loss of vision in the left eye. No headache. General history negative. There was a large central scotoma in the left eye, the peripheral form fields were normal, but there was a definite central bi-temporal hemianopsia for color. She was referred to her family physician and glandular therapy advised. A letter from her under date of April 17, 1927, states, "I cannot see to read with the left eye when the other one is closed because the left blurs over. The other seems all right. Shortly after using the medicine I found an improvement. This continued and I did not have to finish the refilling." Would call this rather uncertain form of pituitary disturbance, apparently improved on glandular feeding.

I am able to report the following interesting and instructive case through the courtesy of Dr. L. Robert Forgrave of St. Joseph, Missouri.

Case 5. C. E., male, age 36, came to me complaining of failing vision and periodic headaches. Family history negative. In February, 1916, patient developed headache lasting about an hour at a time, these were accompanied by vomiting, and recurred every three or four weeks for nine months, after which they increased in violence and frequency, until in July, 1918, they were recurring once a week. About this time the sight of the right eye commenced to fail, and in about fifteen days the left eye also commenced to fail, which continued to progress until December 1, 1918. Patient had to quit work on account of reduced vision. In October 1918, patient was at Mayo Clinic, where he had a spinal puncture, and was told he had evidence of a brain tumor. Blood Wassermann negative, and reflexes normal. Physical examination gave classical syndrome of acromegaly. X-ray showed a very large sella 4 C.M. by 2 C.M. deep, with complete absorption of clinoids and dorsum sella. Vision was perception of light in each eye, with complete bi-temporal hemianopsia. There was a beginning optic atrophy in both eyes. Diagnosis: pituitary tumor with hyper-pituitarism. Sellar decompression was advised and refused. Treatment consisted of five grain doses of thyroid gland, given three times a day. Patient made no progress for two weeks, symptoms remaining stationery. At the end of that time sight and general condition was some improved, and continued to improve steadily, until on January 24, 1919, he was able to resume his duties as express clerk, after which he was not seen until February 20, 1921, he had greatly improved in both general condition and vision. His acromegalic symptoms had decreased

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to a marked degree, his hands and feet were smaller, and the features also much reduced. He still had some cervical kyphosis, enlarged frontals, and prominence of lower jaw, also his hands and feet were not yet normal. There was no evidence of beginning hypopituitarism. The vision in his right eye was 20/20, he had no hemianopia, the color field of the right eye was practically limited to the macula for red and green. In the left eye it was very much reduced for red and practically limited to macula for green. The right optic nerve showed distinct atrophic changes while the left appears normal. This case is of interest, as it demonstrates the apparent benefit of glandular therapy in such cases. April 18, 1927. Have seen this man within the last six months. He is still in excellent condition and is able to carry on his work as express clerk. However, he takes a course of thyroid whenever he begins to feel badly.

Case 6. Mr. E. M., age 58. Consulted us October 27, 1926, for correction of visual disturbance. With correction the vision of the right eye was 20/30, left eye 2/200. There were a number of small white spots in and around the left fovea, but not sufficient to account for reduced vision. Right fundus normal. The right visual field was normal for form and color. The left a decided temporal slant for form with a relative scotoma for small test objects and a nearly complete temporal hemianopsia for color. Sella reported by radiologist as "very large with posterior clinoids absent." A decompression operation was advised but refused so glandular therapy was instituted. He later consulted a very reliable neurological surgeon who confirmed the diagnosis and urged operative interference which was again refused and glandular feeding continued. In a letter dated May 8, 1927, he says he is about the same or perhaps a little better. He would like to try X-ray therapy but says he will have the operation if his vision should get any worse.

Case 7. 62190. Mrs. H. L., age 40. Was referred to us May 11, 1927. She has had headaches for years but worse the last six weeks. Migrainous in type. She has had an appendectomy and partial ovariectomy. Headaches described as terrific for a week before onset of menstruation. No skeletal changes. Vision improved to 20/20-1 each eye. Blurry at times but not hemianopic. Perimeter shows a questionable temporal slant each eye. Fundus normal each eye. X-ray shows sella average size, 12 by 7 mm. with anterior and posterior clinoids nearly meeting. History strongly suggests that headaches may be due as suggested by Kast (35) to physiological enlargement of the pituitary in a closed type of sella associated with the menstrual periods. Acting on this she was given small doses of thyroid and whole gland extract the week preceding menstruation and asked to report, but the time has been too short to learn the result of the treatment.

In this group treated by glandular therapy, only one (case 3) continued to fail and in one case (7) the period of treatment has been too short to justify an opinion. Two (cases 2 and 6) are no worse, possibly slightly improved, while three cases (1, 4 and 5) were definitely improved. An encouraging proportion which certainly justifies this type of therapy where the symptoms are not rapidly progressive, or where operation is contraindicated or refused.

GROUP II.

Case 8. 40340. Mrs. W. J. H., age 50. Consulted us July 26, 1924, complaining of black spots before the eyes. Vision 20/100-1 in the right and 20/100 in the left improved to 20/30 and 20/40 respectively. The ophthalmoscopic examination showed both optic discs slightly light in appearance but probably normal. The mother of eight children, she began to gain in weight at about thirty years of age but nothing in history or general appearance to indicate disturbance of pituitary function. At this time she had a definite bi-temporal decrease in her form field with a definite temporal hemianopsia for color. X-ray reports state the posterior wall of sella almost entirely gone. The possibilities of X-ray and thyroid glandular treatment were explained and

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she was given a grain of thyroid extract twice a day and referred to Dr. Overgaard for radiation therapy. Last seen on October 10, 1924, when her vision with correction was 20/40 in each eye, not much change in field. Complained of having had very severe headache week before. Patient disappeared at this time and subsequent history learned March 31, 1927. Her vision at this time was 14/70 in the right, 2/200 in the left. Vision of right eye improved with correction to 14/30+. She stated that she took twelve treatments from Dr. Overgaard during which time her vision was steadily worse until she could barely see to go about. She then consulted another oculist, a very conservative man, who hospitalized her and advised her to see Dr. Adson of Rochester for operation, which she refused. While in the hospital, she was advised by a patient in a neighboring bed to consult a chiropractor which she promptly did on getting out of the hospital and to her great satisfaction she "began to improve within a week after beginning the treatment." She took 125 adjustments in all at an average of about $1.00 apiece, and assured me she was going to take some more as soon as she could get the money. It was useless to explain that the improvement was undoubtedly due to the delayed action of the X-ray. She was very comfortable and contented although with a definite bi-temporal hemianopsia with contraction of the lower nasal quadrants. She was urged to keep in touch with an oculist for a checking up of fields and vision even though she kept on with the chiropractic adjustments. I think it is reasonable to assume in view of the fact that her vision and fields were changing rather rapidly and the amount of glandular therapy was very small at best, that we can attribute the retention of vision and nearly stationary condition of the fields to the radiation treatment which was given. Although all the credit goes to the chiropractor as she is sure that none of the doctors did her any good, we have the satisfaction of knowing that she would in all probability have been blind by this time had it not been for the radiation therapy.

Case 9. 34134. Miss E. S., age 17. Consulted us October, 1923. Vision reduced to 20/70 in each eye without correction; with correction 20/30 and 20/40. Fields hemianopic on right side with complete hemianopsia on left. Ophthalmoscopic examination normal each eye. No special headache. Has grown very fast, especially her hands and feet. Began menstruating at the age of 12 but soon stopped and has not reappeared. X-ray shows large sella with anterior clinoids indistinct. Dorsum sella long and narrow, the floor shows enlargement. She was referred to her local oculist who in June, 1924, reported condition about the same. At this time we suggested that X-ray or radium be tried. Her physician reported that the patient died February, 1925. Were unable to learn whether the advised treatment had been carried out or not.

Case 10. 30773. Mr. E. A., age 65. Consulted us June 26, 1923. History of vision failing in the left eye for about four months. Vision right eye 20/20, left eye counting fingers at nasal side at four inches. At first examination some nasal contraction of right nasal field, complete left temporal hemianopsia. Fields suggested left homonymous type. Right disc normal, left atrophic. X-ray shows clinoid processes illy defined, sella apparently not enlarged, floor somewhat depressed. He was put on a half grain of thyroid extract twice a day and five grains of whole pituitary extract twice a day. Three months later slight improvement in vision, operation suggested but refused. February, 1925, vision right eye 20/70, left eye blind for over a year. Has completely lost sense of smell, no change in sella since last examination. Radium treatment advised. Two applications by Dr. Quigley. Examination May, 1925, well marked atrophy right optic nerve, left eye atrophy complete. Large doses of thyroid and pituitrin advised up to fifteen grains of each three times a day. June 2, 1925, his physician wrote that his vision is considerably worse. He shows some toxic effect from thyroid so this discontinued. At last examination right field incomplete, nasal heminaopsia. Left eye blind. May 10, 1927, his physician writes that he is practically

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blind. Had been told by an excellent neurological surgeon that an operation would be of no benefit. He is not taking any treatment at this time.

Case 11. 40230. Mrs. E. H. A., age 49. On July 22, 1924, consulted us as she had been told by an oculist in San Francisco she had atrophy of the optic nerve. Vision in the right eye equalled counting fingers at one foot in the lower inner field, left eye 20/40. Left eye had been failing for the last few months. She thought her visual fields were worse during the last month. In the right eye the vision was reduced to an island considerably less than half of the nasal field and in the left eye there was a definite temporal contraction in the form field with the red reduced to a hemianopic area with a quadrant defect below. The right optic disc was pale, possibly beginning atrophy. There was a marked physiological excavation of the left disc which was suspiciously light in color. The X-ray showed absence of both anterior and posterior clinoids, sella practically obliterated. There was no evidence of skeletal disturbance. Menopause which came on gradually was complete at the age of thirty-eight. She was put on thyroid extract, later whole gland pituitary extract combined with the thyroid. She was also given pituitrin sub-cutaneously. On September 19, 1924, she came in complaining of seeing a blue spot all the time before either eye, more pronounced in the left. At out last examination which was October 7, 1924, vision of the right eye was recognition of shadows, left eye with correction 20/30—. There was no change in the fundus appearance from the previous examination. There was a slight increase in the form and color field in the left eye with the nasal field in the right eye slightly larger. She was advised to use radium or X-ray but she eventually returned to her home on the west coast and we did not see her again and have no way of knowing her present condition. From the fact that the form and color fields were somewhat larger at our last examination, it might justify us in the conclusion that the glandular therapy was of some value although in the absence of later information this cannot be stated with certainty.

Case 12. 42904. Mrs. G. P., age 40. Consulted us November 5, 1921, with the history that in January, 1921, during pregnancy, her vision became very poor. For a time she could scarcely see across the table. Vision improved after confinement. Has noticed present trouble for last two or three months. Vision right eye 20/20—, left eye 20/100, not improved. Both optic discs rather pale, left slightly the worse. X-ray report (Dr. N. C. Prince) shows sella decidedly enlarged, both anterior and posterior clinoids ragged, partly destroyed. Form fields show definite bi-temporal hemianopsia with large quadrant nasal defect in lower part of left field. X-ray or radium treatment advised. In January, 1925, she wrote that she was discouraged about the radium but was advised to take another treatment at least. At our last examination which was on June 25, 1925, vision was 20/25+2 in the right eye and 20/100 in the left. Ophthalmoscopic findings the same as at first examination. Up to March 31, 1925, she had had three radium treatments, three X-ray treatments, and at the same time she was given thyroid extract (one-fourth grain) three times a day. In June, 1925, she was put on mixed gland extract (female No. 2, Burroughs-Welcome Company). She was asked to come in later for a check-up on her fields but so far has not done so, although the field examination of June, 1925, showed no change for better or worse from the first examination. In a letter dated May 15, 1927, she states, "I think my eyes are about the same. The best eye is just about normal and there is no improvement in the other."

This case evidently was originally a hyperplasia secondary to pregnancy, later developing either an adenomatous or cystic change. Conditions during observation not affected apparently one way or the other by either radiation or gland treatment, although the fact that she got no worse might indicate that the treatment was of some value.

This group treated by glandular feeding and radiation is less encouraging. Two cases (8 and 12) retained useful vision but were no better than at the

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beginning of treatment. One of these (case 8) definitely worse immediately following the radiation but subsequently improved. One case (10) worse in spite of treatment. One slightly improved (case 11) and one died (case 9). We were unable to learn the cause of death or of her previous visual condition.

GROUP III.

Case 13. 10325. Mrs. R. C., age 21. First consulted us November 11, 1925, with a history that about two weeks previously she noticed she did not see so well with her left eye and had frequent headaches. Vision was 20/50 in the right eye, and ability to see the right-hand letter in five lines of the Snellen chart but no more. The optic discs were light but not necessarily pathologic. The X-ray showed the sella considerably enlarged. By perimetric tests the right field was normal, left relatively hemianopic. She was put on glandular therapy and by December 9, the vision was 20/20—1 in the right eye, and 20/20 in the left. Both fields were normal by January 6, 1926. May 21, 1926, she returned with a vision of 20/70 in each eye. She stated the sudden decrease of vision had come on following acute ptomaine poisoning and in view of her previously normal field and vision, we thought possibly the reduction of vision might be toxic rather than pituitary in origin. General and glandular treatment was continued and by June 11 her vision was back to normal and by June 23 her fields were again practically normal. No further disturbance was noted until the 27th of September, 1926, when she woke with her vision very much reduced. She was very nauseated and within twenty-four hours had no light perception in either eye. Through a misunderstanding she was not seen until September 30 when she was entirely without light perception although the fundus examination was negative. Dr. Keegan saw her in consultation and advised immediate operation, which was done. A trans-nasal approach was selected on account of the enlarged pituitary fossa and the persisting clinoids. The sphenoidal roof was opened with chisel, exposing a moderately tense dural capsule. The dura was incised and a greyish tissue exuded. This tissue was further removed with currette, leaving an apparently empty fossa which was swabbed out with one-fourth strength tincture of iodine. Recovery was uneventful and complete. Within twelve hours she could distinguish form and in three days was able to read. October 7, one week after operation, vision was 20/20—1 in each eye. October 22, three weeks after the operation, both fields were restored to normal and have remained so to date, although reduced glandular feeding is still continued. Patient has resumed her former occupation as teacher, is perfectly healthy, and has had no return of symptoms.

Case 14. 6391. Mrs. H. W. B., age 53. Consulted us February, 1921, for refraction at which time she showed a very moderate hyperopic astigmatism with presbyopia. At this time the only fundus pathology noted was that the arteries indented the veins rather markedly. She returned in July, 1923, with the vision reduced to counting fingers at three feet in the right eye, 20/70 in the left improved to 20/50 with correction. There was nothing ophthalmoscopically to account for the reduced vision in the right eye. Form fields were practically normal, but there was a marked hemianopic recession of the color fields. X-ray report was that the sella was large, 20 by 20 millimeters, with atrophy of the clinoids and the floor of the sella pushed down into the sphenoid area. There were no skeletal changes. She was given a half grain of thyroid extract twice a day and in November she thought her vision was improved. Glandular therapy continued. She returned in March, 1927, with vision much reduced, and was operated on the 12th by Dr. Keegan. Trans-septal approach. The floor of the sella was pushed down and was of paper thinness. About one c.c. of tumor tissue was removed. Almost no bleeding. Cavity swabbed out with one-half strength iodine. There were no complications and the patient made an uneventful recovery. The pathologist reported it as a pituitary adenoma. May 7, 1927, vision with correction, right eye 3/200, left eye 20/100. Nerves rather pale.

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Right more than left. Temporal fields improved. Thyroid and pituitary extract continued.

Case 15. 31147. Mr. R. E. M., age 44. Consulted us first for refraction, January 20, 1911, at which time there was no evidence of any ocular disturbance except a very slight degree of myopic astigmatism with beginning presbyopia. An examination in 1916 mentioned that the right nerve-head seemed a little pale. Perimetric measurements taken at that time showed the form fields normal with a rather definite concentric contraction for blue. The right eye turned in slightly but there was no diplopia. Gave a history of having been cross-eyed at times since childhood. X-ray showed a large sella which was confirmed by a second raying. He was seen again in 1918, when the vision of the right eye equalled counting fingers at fifteen inches, in the left eye 20/200+1. At this time a blood Wassermann was two-plus with a negative spinal. Ophthalmoscopic examination showed the right nerve-head very pale, left moderately so. Fields showed no central scotoma, blind spots normal, has upper temporal slant, especially for red. Had been taking thyroid extract for two years and was beginning to show evidence of thyroid irritation as shown by nervousness and rapid pulse. Thyroid medication stopped for two months. In August, 1924, was advised in addition to the thyroid to try X-ray treatment and told that an operation would probably be necessary. August 26, 1924, referred to Dr. J. J. Keegan for operation, who exposed the tumor by the right trans-frontal route. He stated that nothing much was left of the right optic nerve except a fibrous cord surrounded by the tumor, left optic nerve was not exposed. Large gelatinous adenoma was curretted from the pituitary fossa. There was no special bleeding or shock but during the next few days the patient complained that he could not see light with either eye. Before operation vision of the left eye was 20/200. At our last examination, which was on January 15, 1925, there was no light perception either eye with both optic discs atrophic.

Case 16. 50155. Mr. G. T. U., age 30. Consulted us October 6, 1925, because his glasses were not satisfactory. Had headaches previous to getting the glasses he is now wearing. Thought his left eye was worse recently. Had noticed both failing for over a year. Could not see well with the left for the last three or four months. Vision right eye 20/70, left eye 20/100. Not improved over the glasses he is wearing. Left eye vision comes and goes. Pupil reactions normal. Felt a drawing sensation on the left side of his face for last two or three weeks. No history of nasal trouble. Marked atrophy of optic discs. Has noticed hemianopic effect for about three months, which came on suddenly. Has a definite left homonymous hemianopsia with a quadrant defect in the lower left nasal field. Radiologist reports sella very large, twice the usual depth and breadth with increase in sphenoid. Through his family physician glandular treatment was instituted, one grain of pituitary extract by mouth three times a day, and one grain of thyroid extract three times a day. Also hypodermic injections of pituitary extract three times a week, sub-cutaneously. Operation to be considered later. At examination, February 27, 1926, vision without correction 10/200 in the right eye and counting fingers in the left. X-ray showed sella turcica very large, posterior clinoid largely destroyed. Wassermann negative. A week later, vision same. Sleepy all the time. Discs decidedly atrophic. Sub-sellar decompression advised. Operation by my colleague, Dr. John B. Potts. Intra-septal operation. Floor of sella very thin. Complete absence of bone in posterior portion. Floor removed, tumor mass excised, no fluid. Patient returned to bed in good condition. Four or five hours later gradually lost consciousness, possibly due to post-operative hemorrhage. Slowly recovered consciousness but lost sight in right eye, which did not return. Mentality remained somewhat cloudy. Movements of left arm and leg not well co-ordinated. Discharged from hospital as bed patient. Vision nil, mentality clouded, physical condition good. Patient passed away August 25, 1926, five and one-half

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months after operation, probably due to intra-dural extension of the tumor mass.

Pre or post-operative glandular therapy was employed in these four cases. One died (case 16) five months post-operative, one (case 15) blind post-operative, one (case 14) definitely improved and the other (case 13) fully restored to normal. Although the glandular therapy was not sufficient to avoid operative interference in this case, I am convinced that it was of value although but for the operation there is every reason to think that the blindness would have been permanent.

GROUP IV.

Case 17. 51292. Mrs. R. L. S., age 41. Consulted us November 30, 1925, complaining of headache, weakness and nervousness. Vision 20/200 right eye, 3/200, slightly better at nasal side, left eye. Optic discs about normal in color. She noticed her hands and feet beginning to enlarge about a year previously and had suffered more or less from grippe during the last six years. The right visual field was normal. The upper left temporal form field was reduced with definitely hemianopic color field. The pituitary fossa at this time was greatly enlarged, measuring 23 mm. by 17 mm. Deep X-ray therapy was advised followed by nausea, vomiting, intense headache and nervousness. This continued to increase until February 2, 1926, when she could only distinguish form and light with the left eye and could read with difficulty with the right eye. By this time she was definitely acromegalic in appearance, large hands and feet, and heavy features. A trans-nasal pituitary decompression was performed by Dr. Keegan on February 6, 1926, the operation being complicated by thickness and density of the bone. The bony covering of the fossa was removed, the dural capsule of the pituitary tumor incised followed by a gush of dark red fluid, evidently degenerated tumor tissue. Further tumor tissue was removed with currette. There was some post-operative bleeding which was easily controlled. The following day the patient noted that the room seemed brighter and she could count fingers at the nasal side which she could not do before the operation. Her headache was entirely relieved. On the third day she was able to read with her right eye followed by rapid improvement in vision and general symptoms. March 3, the vision was 20/20 in the right eye, and 20/200 in the left with decided improvement in the visual field. Several months later she reported to Dr. Keegan, "I sew, read, etc., and have but very few headaches, sleep well and am in every way more normal. My looks are also improving."

Case 18. 3865. Mrs. D. T., age 40. Referred to us November 22, 1920, on account of blurred vision. Right eye counts fingers at ten inches at the nasal side, left eye 20/100. Pupil reactions poor. Glasses do not help. Right disc normal in appearance, left disc markedly atrophic. Fields nearly symmetrical with a large lower quadrant nasal defect in both. X-ray showed sella 12 mm. long, 6 mm. deep, without loss of clinoids. Bridge type of sella. The appearance suggested a super-sellar type of tumor (Wick). Pupils react sluggishly to light but promptly to accommodation. General physical and serological examination was negative. She was returned to her local oculist for observation. Re-examined March 9, 1921, at which time the vision of her right eye was counting fingers, left eye 20/200. The use of radium or a sub-temporal decompression was suggested in a letter to her oculist. An examination of her field showed a slight improvement over the one taken at her first visit. A letter from Dr. Adson (Rochester, Minn., April 12, 1921) stated that he found a super-sellar tumor apparently not involving the pituitary. This was removed through a left trans-frontal approach followed by three massive doses of radium. The tumor was reported as a granular endothelioma. May 6, 1927, her oculist, Dr. Nelson, of Norfolk, Nebraska, writes that at the present time, six and one-half years after operation, her vision in right eye, no light perception, left eye 20/20, with slight contraction of her temporal field. Her general health is good with no evidence of return of the tumor.

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Post-operative radiation was employed in both these cases. They each have normal vision in one eye. Vision is reduced in the other. There has been no return of symptoms after one and six years respectively. While the value of radiation cannot be proven, it is reasonable to believe that it was of value.

GROUP V.

Case 19. 36136. Chas. H., age 23. Was referred to us January, 1924, by Dr. A. D. Dunn, who reported that he had lost the vision of his left eye in the last five or six weeks. Vision right eye 20/25, left eye shadows at the nasal side. The right optic disc was slightly pale, left decidedly atrophic. Fields at this time, right eye, temporal hemianopsia for red, with marked reduction of the form field; left eye, temporal hemianopsia with loss of fixation. X-ray showed enlargement of the pituitary fossa with loss of the clinoid processes. On February 4, 1926, a trans-frontal operation from the left side was performed by Dr. Keegan disclosing a tumor mass bulging between the optic nerves in front. The incision was followed by a gush of cystic fluid after which considerable reddish tumor tissue was curretted from the cavity beneath the chiasm. There were no post-operative complications and on February 18, 1924, two weeks after the operation, the vision of the right eye was 20/15—, left eye 20/70—, with almost complete restoration of the fields. His condition continued satisfactory until May, 1925, when he reported on the 8th of that month with his vision reduced to 20/40+3 in the right, 20/40—1 in the left, fields reduced to an incomplete temporal hemianopsia on the right, almost complete on the left. He had been having severe pains in his head, the fundus showed a slight pallor of the right disc, more than the left. Dr. Keegan advised a second operation and on May 11 approached the hypophysis from the right frontal region. A dense mass was again found in front of the optic chiasm. This was incised and curretted thoroughly with no post-operative complications. He was discharged May 25 and received a course of X-ray treatment with pituitary and thyroid extract in the intervals. At our last examination, January 10, 1927, twenty months after the second operation, he had a vision of 20/15—in the right eye, 20/20—in the left, with practically normal fields in both eyes. Just how much the post-operative use of radiation and glandular therapy had to do with the final outcome, cannot be determined, but it would seem from the result that this follow-up treatment was at least worth while.

Case 20. 15544. Mrs. L. E. H., age 44. Consulted us July 12, 1915, on account of failing vision and partial paresis of left internal rectus. Ten years previous to July, 1915, began to see double and for several years had quite a large defect in the right visual field which she thought was somewhat better than previously, large physiological excavation of optic discs, tension normal. Fields in the right eye showed a definite vertical hemianopsia, left eye large quadrant defect in the upper outer field. She was able to tolerate 300 grams of glucose without glycoceria but became very stupid after taking this amount. Was not entirely unconscious, could be aroused, but would drop off to sleep again at once. Vomited a number of times. This came on eighteen hours after taking the glucose. Renal secretions much reduced without showing sugar or albumin. She began to improve promptly on the use of pituitrin. Re-examination November 12, 1919, vision with correction 20/20+5, left eye 20/30—. Reported had very severe headache before using pituitary extract but not since. Had not used any extract for a year or two. Feels well but loses place in reading more than formerly. Physiological excavation same but margins atrophic. April, 1920, vision 20/20 with corrections each eye. Takes pituitary extract regularly but is sure improvement is due to osteopathic treatment. Re-examined November 20, 1921, vision 20/40 and 20/70—1; with correction 20/20—3, left 20/50—1. Is now having headaches in back of head since severe sick spell (character not known) last spring. Started with vomiting and terrible pain in temporal region. X-ray shows anterior clinoid present and almost normal. Posterior clinoid and floor

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of sella absorbed, pituitary extending into the sphenoid cell. Radiographic diagnosis, pituitary tumor. Owing to the increase of symptoms, a sub-sellar decompression was advised and performed by Dr. J. B. Potts, November 22, 1921. An opening ¾ by ⅝ of an inch in diameter was made in the floor of the sella followed by a considerable flow of fluid. There was no evidence of dural tension following the decompression. Patient had no reaction post-operative but on the fifth day developed acute mania which subsided in twenty-four hours. Paresis of the left internal rectus was relieved by the sixth day, with return of the lower temporal right field and full temporal field in the left. A post-operative X-ray report stated that the clinoids are visible, the posterior wall of the sphenoids reduced to a remnant, the floor of the sphenoid and sella seeming to coincide. A re-examination January, 1923, vision 20/50+2 in the right eye, 20/70 in the left, corrected to 20/20 in the right, 20/50 in the left. Advised to continue thyroid and pituitary treatment. November, 1924, she reported vision a little worse but with correction 20/20 in the right and 20/50 in the left. Perimeter, nearly complete bi-temporal hemianopsia for colors. There were large formfield defects in each eye. She complained of pain in the back of her head after laying on her back but not nearly so severe as before operation.

Case 21. 37915. Mr. R. A. C., age 30. Consulted us April 17, 1924, with a history of failing vision for a year but worse during last six months. Vision 20/50 in the right eye, 6/200 in the left, improved to 20/30—1 with correction in the right eye. Left eye glasses of no benefit. Fundus examination showed rather sharply defined physiological excavation in each eye, somewhat larger and lighter in apperance in the left. There was sharply defined bi-temporal hemianopsia with a lower right quadrant defect. X-ray showed nearly complete destruction of the posterior clinoids, marked enlargement of sella with obliteration of all land marks wtih the exception of the anterior clinoids. The anterior wall was pushed down into the sphenoid cells, obliterating about a third of each cell. Physical examination showed rather tapering fingers, feet enlarging during the last five years, libido decidedly less during the same period. He feels he is not so active either mentally or physically during the past five months. No change in weight previous to last year, has gained fifty pounds in the last 10 to 12 months. No increase in height or size of head as indicated by hat size. Fairly heavy mustache, otherwise hair on face scanty. Skin fair. Blonde type. Has two healthy children, youngest four years old. Has had considerable headache through eyes and back of neck. Operation suggested but refused so referred to Dr. Overgaard for X-ray treatment and put on increasing doses of thyroid extract up to one grain twice a day along with pituitary extract. Re-examination May 26, vision and fields practically the same. Radiation treatment continued. June 19, wrote that about a week previously after going to sleep had a sensation as though something burst in his head. Next morning had terrific headache which gradully disappeared. September 24 wrote that he was about the same. Has lost considerable weight. On October 24 vision failing. As his home is a considerable distance, he was advised to keep in touch with some one nearer home. A letter from his wife dated May, 1926, stated that he had died a few days before following an operation by one of our very excellent neurological surgeons, but did not state the character of the operation or any condition found.

Case 22. 30341. Mrs. J. W., age 30. Had been under our observation since January, 1913, at which time she consulted us on account of symptoms incident to errors of refraction and muscle unbalance. Nothing at this time suggestive of any intra-cranial disturbance except that a year before she had severe pressure headaches during latter weeks of pregnancy. She was seen at various times but in June, 1923, gave the history that about two years previously had noticed vision blurring. After confinement, November 16, 1921, vision failed in her left eye. She had a "nervous breakdown," was told by a competent neurologist that the trouble was possibly due to high

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blood pressure. She stated that everything had a glassy look although the vision in her right eye was 20/15—3, no light perception in the left. The right eye was normal on ophthalmoscopic examination, left eye complete optic atrophy. No record of fields or X-ray was taken at this time, but in January, 1924, six months later, vision was 20/20+ in the right eye and ability to count fingers at eighteen inches at the temporal side in the left. The right eye showed a decided temporal quadrant defect for color with the field of the left eye reduced to a small area in the nasal half. X-ray practically negative so far as sella was concerned. She was referred to her family physician with the suggestion that the symptoms might be due to a slight enlargement of the hypophysis exerting pressure on the chiasm and possibly secondary to her last pregnancy. A guarded prognosis for improvement was given. Glandular feeding was advised to be continued for at least six months. She reported frequently either personally or by letter and although at times she seemed to be slightly improved, it was her general impression that she was losing ground. On May 3, 1926, a left trans-frontal operation was performed by Dr. Keegan, who found a firm slightly movable encapsulated mass about one cenitmeter in diameter, extending to the left from the pituitary. Owing to the firm attachment of the base and intimate relation to adjacent structures it was inadvisable to remove all of the mass. Recovery was uneventful. The pathologist, Dr. Niehaus, reports, "large cells, with vascular nucleus, arranged in cords and whorls. Shape usually rounded or oval. Diagnosis, carcinoma of anterior lobe." She was given post-operative treatment with radium and X-ray but with very doubtful prognosis as to improvement until June 1, 1926, when the patient thought the vision was improving each day. The last examination which was September 10, 1926, vision in the right eye was 20/30+1, left eye no light perception. The right optic disc looked practically normal. There was still a large quadrant defect in the upper temporal field. A letter from her husband dated May 9, 1927, states that her condition is about the same as before the operation. Has taken no medicine for over three years, and is considering a second operation, but hesitates to have it done.

Case 23, 52384—Mrs. C. A. C. Age 48. Consulted us January 30, 1926. Thought her eyes were failing the last few weeks. Vision right eye 10/200, left eye 20/70. Not improved. She had noted poor vision in her right eye for three months. No history of headaches or pain in the eyes. Both optic nerves were pale, especially the right with shallow cupping but tension normal. X-ray showed sella small but within normal limits. Visual field showed some reduction of right form field but almost complete hemianopsia for red, left field normal. She was given potassium iodide and focal infections corrected. She returned March 17, 1926, when her vision was found to be 5/200 on the right and 4/200 on the left with the fields reduced to a bi-temporal hemianopsia with a large quadrant defect in the lower nasal field. March 19, Dr. Keegan exposed the hypophyseal region by a right trans-frontal operation. Quoting his report "a lobulated grayish translucent tumor mass was seen beneath the right optic nerve. The very thin capsule was incised and a considerable amount of the tumor tissue curretted out beneath the optic chiasm. There was little bleeding. The dura was closed and the bone flap replaced. There was almost no post-operative reaction, vision was noticeably improved on the fourth day and central vision returned to normal within a week. The fields were completely normal for both form and color on April 12." We have no further record of the patient until April, 1927, when a letter from her family physician, Dr. Sullenworth of Charles City, Iowa, said that in the past few months there seemed to be a cloud over the temporal half of the right visual field. He enclosed a chart showing a temporal hemianopsia of the right eye and a rather concentric contraction of the form field in the left. We asked her to report, which she did on April 16, at which time the vision in the right eye was 9/200, left eye 20/40-2 with very irregular form and color fields which varied from

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day to day. She was seen by Dr. Keegan who advised against further operation at this time. She was given radiation treatment and thyroid extract, one-half grain three times a day. The patient was quite sure that her vision was worse after the X-ray treatment and there may have been a slight contraction of the visual fields although this was rather doubtful, as her fields varied considerably from day to day, but on May 3 the vision in the right eye was 20/70, left eye 20/21-1. No improvement in the visual fields. The patient was very nervous and dissatisfied and insisted on another operation. On May 9, 1927, Dr. Keegan approached from the left side and found a solid mass beneath and back of the chiasm. This was curretted away and the wound closed. No post-operative complications. The pathologist reported the type cells as large and oblong. No cell division noted. Appearance suggests malignancy with a tentative diagnosis of sarcoma of the pituitary. May 25, 1927, vision right eye 10/70, left eye 10/40-1. Outer quadrant of each disc pale. Otherwise fundi normal. Both fields definitely larger than before operation. General condition excellent.

The combined use of glandular therapy, radiation, and operation in this group resulted in staying the process or improvement in four of the five and the lack of success in Case 21, who died shortly post-operative, can be attributed more to unforeseen complications than to incorrect treatment.

GROUP VI.

Case 24, 58860—Mr. J. S. Age 29. Consulted us November, 1926, on account of reduced vision. Had been told he had optic atrophy. Stated could see fairly well with the right eye until a week ago. Left eye nearly blind for a year. No venereal history. Wassermann negative. X-ray showed the sella very large, no clinoids demonstrable. Fields showed small quadrant of vision up and in right eye including fixation point, left eye blind. Operative prognosis discussed with patient, but he decided to take the chance and was referred to Dr. Keegan, who opened the sella by the trans-nasal route on November 17. The sphenoidal wall was removed with some difficulty. The dura was uncovered over an area about one centimeter in diameter. This was incised crucially and contents of pituitary fossa curretted. Soft grayish tumor was removed, appearing like cortical tissue. Nothing further was done. There was pressure tending to exude the tumor mass. There was some question as to the location of the opening, although landmarks and vision were entirely satisfactory. A small amount of clear fluid, possibly cerebral spinal, was seen escaping. There was a little bleeding. Fossa was sponged with tincture of iodine. No post-operative complications until the third night, post-operative, when he developed a severe headache radiating to the back of the neck. Chill, temperature 103.4. Neck slightly rigid. He continued with severe irritation until the sixth day post-operative after which he made an uneventful recovery. Our last examination was January 4, 1927, at which time the vision of his right eye was 20/200, left eye hand movements. Both optic discs very light. Fields slightly larger than at first examination. May 15, 1927, reports, "Eyes seem some better. No headache, but nervous at times. Can see well at distance, but not so well for close."

Case 25, 39757—Mrs. G. B. P. Age 30. July 3, 1924. Noticed poor vision in the right eye last two weeks. Had headaches and pain back of the eyes. Vision 20/50-1 in the right, 5/200 in the left. Both optic discs pale. Considerable nose and throat pathology. Two years ago noticed sudden cesation of menstruation which did not return. X-ray showed a moderate sellar enlargement, posterior clinoids gone, tumor mass apparently shoved forward obliterating about two-thirds of the sphenoid cells. There was almost complete symmetrical bi-temporal hemianopsia for form, complete for blue. The seriousness of her situation was carefully explained, treatment urged, but the husband was very averse to having anything done and insisted on treating her with Christian Science, which was done for about a year. In the meantime she became totally blind and almost unmanageable. Finally, ac-

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cording to reports of neighbors, in self defense he took her to Dr. Cushing at Boston in July, 1926, who operated by the trans-nasal route. Husband will not allow examination of patient, but says general condition is greatly improved since the operation, but that she is quite blind and has to be lead whenever she leaves her home. Those who have seen her say she is developing the typical physical characteristics of acromegaly.

Case 26, 25366—Mr. G. W. Age 49. November 16, 1922. Complained of failing vision which he thought was getting worse. Vision right eye 2/200, nasal side; left eye 20/50-1. Pupillary reflexes present, but slow. Both optic discs pale. Both upper outer fields defective. Large quadrant defect, lower outer right field. Anterior clinoids were eroded. Radiograph shows pituitary body enlarged forcing the floor of the sella into the sphenoid cells. September 16, 1922, was operated by Dr. Adson of Rochester by trans-frontal route, exposing a round dark colored encapsulated tumor separating the two optic nerves and pressing the optic chiasm backward. Tumor was slit and contents shelled out. No special post-operative inconvenience. Re-examined by us December 6, 1922, when with the right eye could see the first letter in each row of the Snellen type down to 20/40, left eye 20/20. Thought that sight was improving. Had no headaches. Unable to get field measurements at this time. Have not heard from him since.

Case 27, 58817—Mr. G. B. Age 37. Consulted us October, 1926. History of vision failing since spring. He could recognize motion at the distance of one foot with the right eye, 20/40-1 with the left, corrected to 20/20. Pupil reaction normal, right fundus normal, except for a few light spots. Left eye normal. General examination negative. X-ray showed the sella wide above with indistinct clinoids. Diagnosed by Dr. Keegan as a cystic tumor of the pituitary and recommended transnasal opening of the cyst. The fields were interesting in that there was a very questionable island of vision in the upper outer quadrant of the right field with a complete temporal hemianopsia in the left. Dr. Keegan performed a transnasal decompression, found the floor of the pituitary fossa dense, the dura was incised with no distinct tumor tissue identified. A clear fluid escaped toward the end of currettage which suggested cerebral spinal fluid. The patient was not a good operative subject, stopped breathing during the course of the operation, requiring artificial respiration. He was immediately relieved of his headaches which had been severe and an examination on October 29, he had a vision of 20/40 in the right eye and 20/50 in the left corrected to 20/30 and 20/20 respectively. The right field was restored to normal with an extensive quadrant defect in the left upper temporal field. He went home with instructions to take a half grain of thyroid extract three times a day and a grain of pituitary whole gland extract three times a day. He returned December 1, 1926, seven weeks post-operative, stating that his headaches had returned four or five days previously and that the vision of his right eye was decidedly worse than it had been. The field now showed a vertical hemianopsia on the right eye with an almost complete temporal hemianopsia of the left. He was sent to the hospital on December 3. Dr. Keegan did a transfrontal operation on the right side, exposing a glandular tumor anteriorly between the optic nerves. Not much tension evident. Evidently cystic in character. The contents were curretted out, leaving a large cavity, beneath the optic chiasm in the pituitary fossa. The operation was entirely satisfactory, but the patent did not recover well from the anesthetic, was irrational and restless, temperature of 105, died two days later, symptoms suggesting acute brain oedema. The autopsy revealed a super-sellar tumor extending up behind the optic chiasma pressing deeply into the floor of the third ventricle, the general intra-cranial picture suggesting evidence of pressure. The pituitary gland was separate from this tumor and was flattened out on the floor of the sella turcica. Pathological report gave a diagnosis of papillary cyst adenoma of pituitary stalk. This case emphasizes the difficulty of making an exact localizing diagnosis of growth and enlargement in this region and

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further emphasizes that a thoroughly skillful and satisfactory operation may be followed by fatal termination. The right vertical hemianopsia might have suggested the location of the tumor.

Operation alone improved two of this group (24 and 26). One is blind through temporizing with cults until complete optic atrophy rendered a technically successful operation unavailing. Case 27 was definitely improved by the first operation and but for an unavoidable surgical complication would in all probability have been further benefited by the second.

GROUP VII.

Case 28—Mr. C. A. H. Age 30. January 8, 1919. Right eye blind, left eye 20/50. Temporal half of left field gone. Both optic discs atrophic. Has marked acromegaly of robust type. Stated that decompression operation had been attempted in 1917, but abandoned on account of hemorrhage. Frazier of Philadelphia had refused to operate, stating it would be of no benefit. Possibilities of operation explained to patient. At first wanted operation, later refused. Report of death received later, but date not given.

Case 29—Mr. B. M. S. Age 67. April 3, 1919. Entirely blind. Radiograph showed a large pituitary tumor involving the entire pituitary region and crowding in on the sphenoids, extending well back of the posterior clinoid. Much more dense than the ordinary findings in pituitary tumor (Dr. Mick). According to the record he was under observation for a week, then returned to his local oculist. Letter from Dr. McPherson under date of May 4, 1927, stated that the patient gradually failed and slowly developed exophthalmos both eyes. Nose completely obstructed, face swollen and bulging and died on the 24th of August, 1919, some four months after our examination. This was evidently a rapidly growing malignancy of the hypophysis which progressed to fatal termination without treatment.

Case 30, 58820—Mrs. H. W. B. Age 50. Consulted us November 9, 1926. Trouble with vision, right eye worse. Vision without correction 20/200 in the right, 20/40— in the left. Right improved to 20/70+, left eye 20/20—. Vision of right eye comes and goes. Right outer quadrant paler than left. Left practically normal. Little or no headache. Noticed a year ago that she could not see the end of words. Radiologist reported sella turcica twenty-five millimeters horizontally, fifteen vertically, with absorption of the posterior clinoid processes. Space normally occupied by the pituitary gland showed several areas of calcification. There was a definite tumor mass involving the sphenoid and ethmoid areas on both sides, partially filling the frontal sinuses, especially on the left side. Markedly acromegalic. She was advised to have herself kept under observation and on May 8, 1927, reported that no change whatever had taken place. She was free from pain or headache, and had taken no treatment of any kind. Another X-ray taken at D. showed the gland enlarged "three times its normal size, exactly as at Omaha."

Two (28 and 29) of these three untreated cases are presented to show the end results where nothing is or can be done. Case 30 is temporizing. The fact that the process is relatively quiescent gives her a false security. Every effort is made to keep her under competent observation, but the outcome is problematical to say the least.

SUMMARY

In this series, the frequency of women (57%) over men (43%) is not as high as is sometimes observed. As would be expected, most (77%) were in the middle decades of life, rather evenly distributed from twenty to sixty years of age. All consulted us on account of visual disturbances with only seven (23%) complaining of headache as an accompanying complaint. In 60% the optic discs were pale or atrophic, but the degree of visual disturbance was often much greater (e. g., Case 13) than indicated by the appearance of the fundus.

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Field changes were characteristic. Sixty-seven per cent showed definite bi-temporal hemianopsia and 10% decided temporal slants. In 7% there was homonymous hemianopsia, while in two cases (7%) there was a tendency toward a bi-nasal defect. In the remainder, vision was too poor for field measurement. Twenty per cent showed evidence of skeletal changes (acromegaly). The estimation of type was necessarily uncertain, except in the operated cases, but judging from symptoms and X-ray findings, 70% were probably primarily intra-sellar, 20% super-sellar, the remainder entirely questionable. Ten (33%) were found at operation to be adenomas or cyst adenomas, while four (13%) had undergone some form of malignant change. Of the seven cases (23%) treated by glandular therapy alone, four were definitely improved, and three not improved or worse. Of five cases (16%) on gland and radiation treatment, one was improved, one the same or worse, one dead, and in two the present condition is unknown. Four (13%) were given gland treatment in addition to operative interference. One was improved, one blind, and one dead. Radiation and operation two cases (7%) each have normal vision in one eye, the other much reduced. Of five (16%) on combined treatment—gland, radiation, and operation—four were the same or improved, while one died (post-operative). Of the four (13%) limited to operation alone, two were definitely improved, one worse, and one dead. Of the remainder, two were unoperable and died. One is under observation not accepting any treatment; prognosis unfavorable.

CONCLUSIONS

The classical signs and symptoms of hypophyseal disturbance are easily recognized. However, unexplained headaches, unusual field and visual changes, and questionable skeletal abnormalities also should warrant a careful consideration of their being of pituitary origin and especially so in women if the above conditions are associated with menstruation, pregnancy, or menopause.

As surgical interference is too often reserved as a last resort it is to be expected that the reports of neurological surgeons favor operative interference. Nevertheless, there are many cases which can be relieved by non-surgical measures and where they are seen early and can be kept under observation, glandular and radiation therapy is of value in many instances.

Operative cases definitely super-sellar should be referred to the general neurological surgeon, but sub-sellar decompressions and currettments can certainly be handled as well or better by the rhinological surgeon who is accustomed to work by reflected light in a narrow field and skilled in septal and sphenoidal surgery.

Cases of hypophyseal disturbance are entitled to our most careful consideration and best efforts and in many instances will respond satisfactorily to therapeutic and operative measures.

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Discussion opened by Dr. Swift, Seattle:

It is a pleasure indeed to have an opportunity to open the discussion of this paper. The members of this Society have been particularly fortunate this year in having the men from the East who are members of the Ophthalmic and Otolaryngological Board and to have them be willing to present such wonderful papers.

This paper of Dr. Patton's covers such a broad field that it is difficult in discussing it not to forget some of the important points. I had the pleasure of reading his paper prior to the meeting and have reread it since coming to Spokane. I can add nothing of value in discussing this paper, but

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would like to emphasize one or two points that are made by Dr. Patton, but perhaps not singled out by him as of great value.

The neurological surgeon must make the final decision as to the advisability of operation and of course must choose the route by which he feels justified in attempting the removal of the tumor. When the tumor has reached the supracellar area, it is very difficult to remove it by the transsphenoidal route.

Tomorrow I will show some specimens in which the tumor had begun in the sella and later extended into the temporo sphenoidal lobes on either side. I am sure no one would attempt to remove such a tumor by the transsphenoidal route. A little point to be remembered in this connection is frequently elicited in the history. A patient states that he has suddenly lost all pain, the symptoms all subside, and at a later date he will state that he again had severe pain. Pain in these tumors is due to pressure against the dura, in the first instance, and in the second instance, a dragging on the branches of the internal carotid, which causes a sympathetic irritation resulting in severe pain. The second pain is sharper and more constant and throbbing than the first pain, which is dural and intermittent.

Another point to be remembered is the importance of having a true pituitary lesion, even though the symptoms of bitemporal hemianopsia and X-ray findings point directly to the location. I have in mind a case referred to me by Dr. Waltz of Seattle in which at autopsy we found an enormous aneurism of the anterior communicating branch of the circle of Willis. This had extended downward and eroded the entire sphenoid and had the appearance of a polyp in the sphenoid. Third nerve involvement associated with pain from the ophthalmic branch of the fifty caused Dr. Waltz to hesitate in making his diagnosis of a polyp and we eventually diagnosed the case prior to death.

A similar case having the eye findings, but not the X-ray findings, proved to be a supracellar aneurism of the circle of Willis which ruptured, causing death. The true diagnosis was not made until autopsy.

I mention these two cases as of importance in differential diagnosis.

Another point that will bear emphasis is that relating to the time of operation. There is no use in operating a case that has a choked disc in the hope of relieving the condition. Even though one should improve conditions for a while, the end result is not satisfactory. It is far better to operate early than to wait until either general edema of the brain has caused a choking of the discs or until mental deterioration has been superimposed upon the already complicated intracranial condition. The best clue is exactly as Dr. Patton has stated, a continued decrease in vision. As soon as one is satisfied of this, it is important to operate.

The value of the X-ray in these cases is still questionable. I do not believe one should attempt to give the X-ray without doing a broad decompression. The mere fact that bone itself throws a shadow is sufficient proof that certain rays are retarded. It stands to reason that better radiation can be accomplished after a decompression. The decompression also assists in relieving the intracranial pressure and when the X-ray is used with full intensity, it prevents the secondary edema from causing trouble.

Dr. Greenwood:

Mr. President and Members of the Society who have so kindly invited us here:

This paper of Dr. Patton's gives me a double opportunity. In the first place, the disease is, for its diagnosis, largely dependent on taking visual fields, and I want to criticize the ophthalmologists of the country, and myself along with them. As a member of the American Board for Ophthalmic Examinations for ten years, off and on, I have found that the one subject that most men fall down on in the examination is perimetry. This is the subject that more men, in taking the examination, are conditioned in than any other, and after study must pass it before one of the Board members later. How

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such men can, with good judgment, take care of cases of glaucoma without a perimeter, I do not know. Occasionally, men in the examination have not ever known how to handle a perimeter of any kind and claim they do not take the fields of vision. Therefore, I want to call your attention this morning to this and to induce you all to take a greater interest in the subject of perimetry. One of your Western confreres told me recently that he took the field in every case of refraction that he did, and by this custom has picked up a great many conditions you and I might not find.

I wish, by citing a few cases, to show the necessity of taking fields. Recently a surgeon operated on a man who came to me a few days before operation, having passed through the hands of six ophthalmologists. None of them had ever taken the fields. He was nearly blind from supposed optic nerve atrophy. He had a negative Wassermann, but had had many courses of antisyphilitic treatment. Examination of fields with a small light showed a complete bi-temporal hemianopsia extending slightly to nasal side. Since his operation for pituitary tumor, his vision has been a little better with perception of movements in the nasal fields.

I have three other cases which have been operated on and all were cases that had been treated for partial atrophy of one nerve and in no case had the field been taken.

Now, the lesson from this paper of Dr. Patton's is that you must not let these patients go blind and you must use your perimeter frequently in your practice. Otherwise, cases fall into the hands of someone who will use it. If you have not got one, get one and when you get it, use it.

Dr. Wilson Johnson, Portland:

At Los Angeles I reported two cases of hypophyseal tumor which I had treated with radium at that time and it is simply a further report of those two cases, both living, one with loss of some vision, both relieved of headaches, except an occasional headache, one about every two weeks, the other not that much. At that time I did not do decompression, but made the application of radium directly under the floor with a device that I use which is made from ordinary applicator and the rays were projected in only one direction. I believe the radium itself produces a dehiscence in the bone and we have a decompression made. Today, I think I would do the decompression and use the radium directing it into the tumor with a radium needle. I think it is a better method than the use of X-ray. It is more direct and offers the patient a chance for relief that is fairly definite in a large proportion of cases not necessarily malignant.

Dr. C. A. Veasey, Spokane:

I wish to express my very great appreciation of the most excellent discussion of this subject which has been given us by Dr. Patton, and to speak of a few points to which he referred.

Dr. Patton stated that all cases of bi-temporal hemianopsia must not be construed to mean hypophyseal tumors. With this statement I am in entire accord. Cases have been reported where vascular conditions have produced absolute or fleeting bi-temporal or the so-called bi-nasal hemianopsia. Some years ago I reported before the American Ophthalmological Society a case of fleeting bi-temporal hemianopsia in which I made repeated examinations over a period of a long time. At one examination the patient would show a complete bi-temporal hemianopsia, and at a succeeding examination it would be found to have disappeared. At other times, there would be a bi-temporal hemiachromatopsia, but the form fields would be intact. Further examinations would show this to have disappeared. The man had marked headaches and both optic nerves were exceedingly pale and partially atrophic. The neurologic symptoms of this case were studied by Dr. William G. Spiller of Philadelphia, and the patient was kept in the hospital for a long time in the hope that, in the event of a fatal issue, an autopsy might be obtained. However, he lived for many years and then disappeared from

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observation, retaining good central vision up to the time of the last observation. It is believed that the symptoms of this case were due to a vascular lesion which might have been an aneurysm or a sclerotic vessel in which the amount of pressure was changed from time to time. The late Dr. Herman Knapp of New York followed a somewhat similar case for several years and succeeded in obtaining an autopsy in which was found a sclerotic vessel pressing on the chiasm.

In regard to the point suggested by Dr. Patton that some of these cases apparently grow worse during treatment, we have recently had under our observation a case of bi-temporal hemianopsia in which the posterior clinoid processes showed marked absorption which grew progressively worse under X-ray treatment.

Dr. Patton (Concluding):

I feel that I have accomplished something worth while with this paper in that it has brought out some valuable discussions. The point emphasized by Dr. Swift with reference to the importance of operating according to the diagnosis is valuable. We get bad results, not so much from not doing the things we should do as from doing the things we should not do. We should make an honest effort through the careful study of visual fields as emphasized by Dr. Greenwood to establish a diagnosis.

Just a word about the use of the perimeter. Some men may be too busy themselves to take visual fields and consequently this valuable diagnostic procedure is neglected. If we cannot take these fields ourselves, we can at least train some bright technician who can take them for us. It does not take so much clinical experience as it does patience and attention to details and you will be gratified to find out how satisfactorily a technician can do this work.

Dr. Veasey emphasized a valuable point. We should use the various agents that we have at hand. A number of our most experienced men have had good results from radiation therapy and from our experience I am convinced of its value. The same may be said of glandular therapy. Some cases are inoperable. Others refuse operative interference. In no instance are we justified in neglecting non-operative therapy where there is a possibility of its being of value to the patient.

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BINOCULAR CORNEAL MICROSCOPY

LAWRENCE G. DUNLAP, M.D., Anaconda, Montana

Just as the Helmholz ophthalmoscope opened the book of fundus pictures to us, so has the Czapski binocular corneal microscope with the aid of the Gullstrand slit lamp and Hencker arm, opened up to us another new field—the anterior segment of the eye with a new appreciation of its anatomy and pathology.

Many years have been spent by ophthalmologists in becoming acquainted with the structural changes which take place in the posterior segment of the eye. Even after constant use of the ophthalmoscope, the literature of ophthalmology is embellished with many new fundus observations, as well as many old ones which are not yet completely understood. Hence, it is unreasonable for us to expect to obtain all the knowledge of the anterior segment of the eye as observed with the slit lamp in a few short hours.

In an effort to be conservative many have tried to keep out of the class of those who are "first to take up the new and last to give up the old." Also many are satisfied with their present method of examination, since they are not conversant with the advantages and value of corneal microscopic examinations, just as many were sure they could practice ophthalmology without fundus examinations when the ophthalmoscope was new.

I trust that the following slides, so kindly loaned me by my friend and teacher, Robert Von der Heydt of Chicago, will arouse your interest in this method of ophthalmic investigation, if it has not already been aroused. Most of these slides are from Vogts Atlas and some few from Von der Heydt's private collection.

Introduction:

No. 1—Path of beam of light through cornea—A C—iris and into vitreous—direction of observation—light temporal.

Cornea:

No. 3—Quadrilateral image forward onto cornea.
Quadrilateral image with narrowed beam (narrow slit aperature).

No. 51 (No. 37 Vogt)—Clearing up stripes of Fuchs in old corneal scar.

No. 53 (No. 43 Vogt)—Honeycombed old corneal scar with Staehli line. (Coloring in Bowman area—probably a crack of Bowmans M.)

No. 37 (No. 49 Vogt)—Corneal nerve fibers. (2) (Steahli line does not show well in slide.) Just show nerve fibers and say they are dichotomously branched and in the anterior half or cornea.

No. 39 (No. 60 Vogt)—Keratoconus—showing endothelial cupping (mosaic of cells). Keratoconus stripes and linear scar repair of cracks (gray broken line).

No. 41 (V. 59)—Kerotocomus—showing thickening of nerve fibers.

No. 43 (V. 61)—Kerotocomus—showing thickening of nerve and white dots on same.

No. 45 (V. 63)—Apex of cone in Keratoconus—rupture of Descemets behind.

No. 49 (V. 68)—Rupture of Descemets in Buphthalmos—Keratoglobus infantile glaucoma or (ox eye).

Iris:

No. 55 (V. 293, colored)—Pupillary pigment collar—irregular width—often wider on top.

No. 57 (V. 321, colored)—Iris Atrophy—the shrinking causing an Ectropion of the uveal (pigment) layer.

No. 59 (V. 301, colored)—Synechia.

No. 99 (V. 289a, colored)—Eye after cataract extraction. Post-operation iris pigment ball traction folds in posterior capsule does not need Needling.

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No. 27 (V. 94)—Shagreen of lens (ant. lens surface reflex under Capsule). (Graining.)
No. 7 (V. 100a)—Beam of light passing through lens showing lamellar surfaces. Vertical ant. Y and post (inverted Y).
No. 9 (V. 100b)—Nuclear surfaces.
Clear (central) internal.
5 and 6—Embryonic (central) nucleus.
4-7—Embryonic (outer) nucleu.
3-8—Senile nucleus.
2-9—Subcapanular layer of separation.
1-10—L. capsule.
No. 11 (V. 127)—Ant. and post Y sutures.
No. 17 (V. 126)—Y suture.
No. 15 (V. 125)—Y suture showing radial fiber direction.
No. 13 (V. 237)—Ant. embryonic nuclear lens spots (very common) (remnants of loose cells in embryonic lens vesicle).
No. 29 (V. 92b)—Lens sutures. } Separation in these give
No. 19 (V. 123)—Lens sutures. } origin to "spokes" in se-
No. 31 (V. 130)—Lens sutures. } nile cataract.
No. 25 (V. 134)—Senile nuclear surface reflections "knobs" rarer type.
No. 23 (V. 138)—Same—more common type.
No. 21 (V. 135)—Same—most typical.
No. 33 (V. 102)—Lens epithelium.
No. 61 (V. 234)—Ant. Capsular cataract.
No. 63 (V. 235a)—Nuclear and lamellar cataract.
No. 65 (V. 213)—Coronary cataract.
No. 67 (New)—Coronary cataract—punctate type.
No. 69 (V. 214, colored)—Blue coronary cataract, Cataracta punctata coerelea.
No. 71 (V. 202b)—Post traumatic posterior rosette shaped (temporary) cataract.
No. 73 (V. 202a)—Partly gone.
No. 75 (V. 260, colored)—Suture cataract—rare—just as green and pretty as picture.
No. 77 (V. 278)—Siderosis—iron carbonate in lens from F. B.
No. 79 (V. 268 a & b)—Cataracta Compilcata (in post. sub-capsular region) as seen by ophthalmoscope.
No. 81 (V. 268)—Same as by *slit lamp*, very typical and correct picture.
No. 83 (V. 210)—Spokes (separation in fibers) in senile cataract.
No. 85 (V. 222)—Senile cataract. Lamellar separation.
No. 87 (V. 216)—Peripheral wedge shaped (senile) cataract changes.
No. 89 (V. 218)—Lamellar separation (ribbing of surface) in laterally dislocated lens.
No. 91 (V. 223)—Lamellar separation (ribbing is pathological).
No. 93 (New)—Ant. Suture (sub-capsular) lens punctation.

Vitreous, etc., Post Capsule.

No. 111 (V. 142)—Vascular (normal remants of embryonic blood supply on post. capsule.
No. 113 (V. 151)—Physiological hyaloid artery remant (common).
No. 115 (New)—Is marked (hyaloid buttons like V. 163).
No. 101 (V. 330)—Vitreous structure (normal).
No. 103 (V. 346)—Normal vitreous structure.
No. 105 (V. 339)—Same.
No. 107 (V. 335)—Fluid (disorganized) vitreous.
No. 108 (V. 356)—Synchisis scintillans—so-called cholesterin crystals in vitreous—now known to be spherical and of a soapy nature.

Discussion opened by Dr. James M. Patton, Omaha:

We are to be congratulated for the privilege of hearing Dr. Dunlap's

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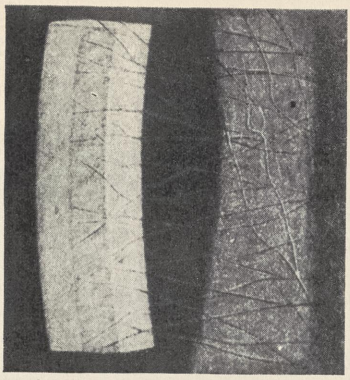

No. 18. Path of beam of light through the cornea illustrating examination under direct and trans-illumination.

No. 293. Pupillary pigment collar, irregular width, often wider at the top.

No. 43. Honey-combed old corneal scar with Steahli line. (Coloring in Bowman area—probably a crack of Bowman's membrane.)

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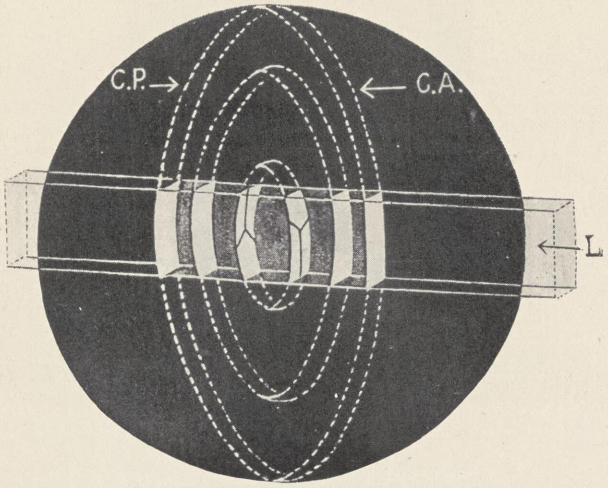
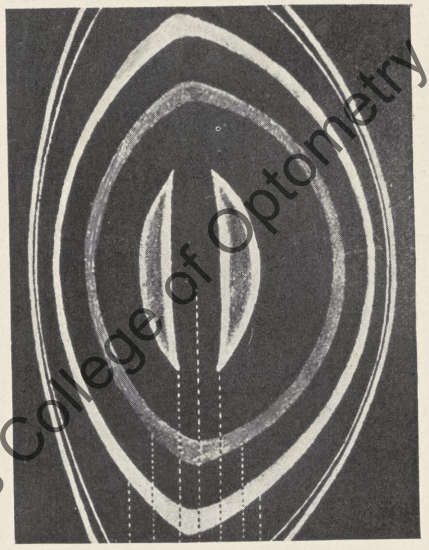


No. 100a. Beam of light passing through lens showing lamellar surfaces. Vertical anterior Y and inverted posterior Y.



No. 100b. Nuclear surfaces. The clear central area, the central and outer embryonic nuclei, the senile nucleus, subcapsular layer of separation and the lens capsule.

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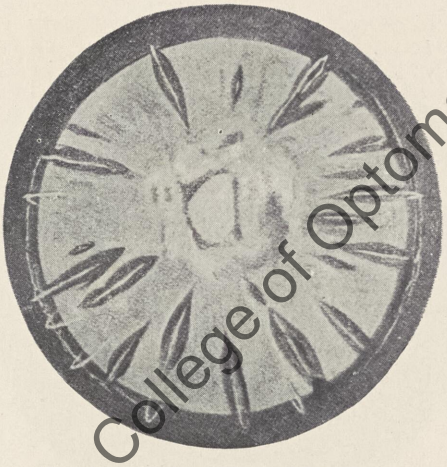
No. 127. Anterior and posterior Y sutures.



No. 235a. Nuclear and lamellar cataract.

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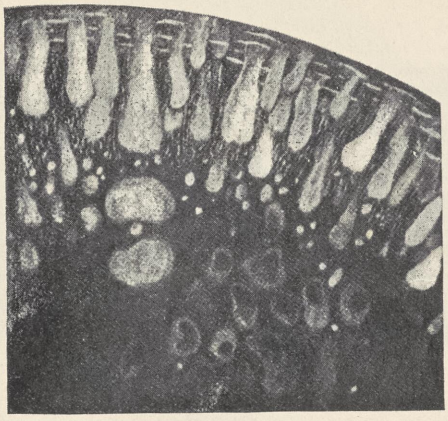
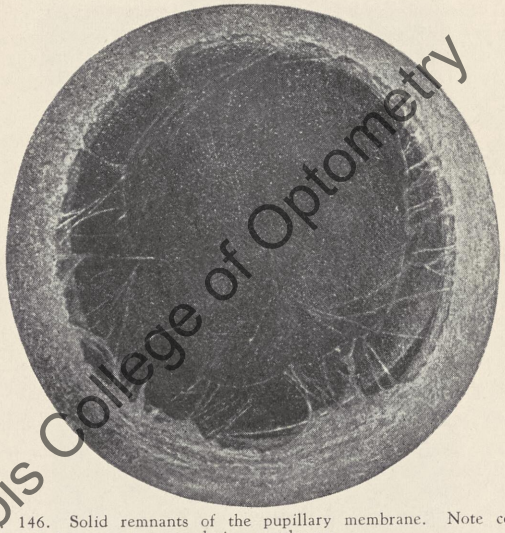


No. 213. Coronary cataract.



No. 146. Solid remnants of the pupillary membrane. Note connection to Iris stroma in contrast to exudative membranes.

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splendid presentation of this recent and important phase of our work. Vital microscopy has not added so much to our actual knowledge of ocular anatomy and pathology as it has given us a refinement of technique and opportunity for observation which were impossible by our former methods. In order to have the advantage of this information, the observer must be willing to devote considerable time both to mastering the technique and to the study of the normal and pathological anatomy as shown by the instrument.

Not infrequently we are asked: "Shall I buy a slit lamp? Is it essential to the practice of ophthalmology? Does it pay?" The answer depends on the individual questioner.

If he is willing to give the time and study necessary to familiarize himself with the instrument and its findings, if he is interested in the refinements of observation and diagnosis and takes pleasure in exploring new fields of research, he will be more than repaid for his outlay of time and money. On the other hand, if he is driven by the demands of a heavy routine practice or if his interests are diverted elsewhere, a slit lamp would prove a poor investment.

Dr. Dunlap has given us an excellent resume of the various fields of usefulness of this method of investigation. It is obvious that the anterior segment of the globe offers the greater opportunities for study and is richest in anatomical detail and pathological change. As the observer becomes more skillful, the posterior segment is well worth investigating and fully rewards painstaking effort.

The general application of vital microscopy has been so fully presented that I will only mention a few instances in which we have found it to be of special value.

A recent case of interstitial corneal staining supposed by the patient to be due to drug instillation, was shown definitely to be secondary to blood pigment. Evidence of changes in the deeper corneal structures in buphthalmos, conical cornea, location of pathology in suspected discaform and interstitial keratitis, and early evidence of marginal or diffuse epithelial dystrophies are well shown by this instrument. The cell content of the aqueous is an indication of uveal involvement, especially sympathetic ophthalmia, some basing their final diagnosis on the slit-lamp findings, while a study of the iris and especially its pigmented border has both diagnostic and prognostic value. Evidence of early changes in the crystalline lenses are best studied by this method and prophylactic measures instituted at this time give the best promise of results.

The slit-lamp amply rewards earnest effort and sharpens for us what has hitherto been a blurred and in some instances a totally indistinct picture.

Dr. Barkan, San Francisco:

I might add to what Dr. Patton has said in answer to the inquiries of the benefits acquired by the purchase of a slit lamp that it would be perhaps well to strike a middle path by acquiring a binocular microscope first without the slit lamp and familiarizing one's self with it, using a lamp such as the Hess Hammer Lamp as the source of illumination—the slit lamp being simply a refinement of the illumination. The use of the microscope without the slit lamp is of much advantage. I think that the slit lamp is very useful and, when purchasing one, should take the whole step and get a big one.

Dr. Stillson, Seattle:

Whether it would be wise to compromise in the matter of getting a slit lamp or hand lamp, the general concensus of opinion after both were tried is that as Dr. Barkan said if you are going to study slit lamp operation, it would be better to give plenty of time and plenty of instruction and go the whole way about getting the proper equipment.

It seemed to me that the views shown by Dr. Dunlap were more or less diagrammatical. Were they the actual reproductions?

Dr. Dunlap: Yes, they are the actual reproductions.

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Dr. Dunlap (Closing):

I am deeply grateful to Dr. Patton for his thorough discussion. In fact, his remarks being more lengthy than mine, remind me of the story told at the banquet last night about the tail wagging the dog. I agree with Dr. Barkan as to the best way to get the most out of the slit lamp. I had a Binocular Corneal Microscopic for almost three years, with just a little arc illumination or various hand lamps as a source of illumination, so that it did not take much time to become proficient in the use of the Gullstrand slit lamp. Now it doesn't take me over five minutes to focus the anterior segment of each eye as a routine examination, and an inestimable amount of good is attained in making sure there is nothing wrong. In answer to Dr. Wilson, I can truthfully say that the anterior segment pictures observed by the slit lamp are just as clear and definite as the Diagrammatic slides shown here today. If I may be allowed one small flight of oratory, I should say that if the ordinary ophthalmologist feeds his body on the proceeds of refractions, he feeds his ophthalmological soul on slit lamp observations.

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EXPERIENCES IN EXTRACTION OF INTRA-OCULAR FOREIGN BODIES

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The above title covers a vast field. It must of necessity be limited. This we shall do by not considering our non-magnetic intraocular foreign bodies, interesting and instructive as the individual cases may have been, and by limiting our consideration to the magnetic ones. As Dr. Hans Barkan reported his experiences with magnetic extractions in the California State Journal in 1920, we will further limit the scope of this paper by restricting ourselves to the magnetic foreign bodies posterior to the plane of the iris since 1920. The chart which I am presenting represents an analysis of 61 magnet extractions since 1920. Statistics are notoriously misleading and, in the case of intraocular foreign bodies, are well nigh useless, because the course of events and ultimate outcome in each case depends so much upon various factors and different combinations, such as initial infection, site of perforation, size and shape of the foreign body, etc., etc. One can however profit much by reviewing one's personal experiences and comparing and correlating them with those of one's colleagues. Indeed one does arrive at certain conclusions and it is surprising how one's views are clarified when studying such a chart as this and passing the cases in review.

Our cases were all done under local anaesthesia with a Haab, giant magnet and Hirshberg hand magnet. I shall not dilate upon the question of magnets in the presence of Dr. Lancaster who will, I trust, take pleasure in enlightening us in the discussion. We operate with the patient sitting in front of the magnet for the anterior route and we value the possibility of immediate retraction of the subject's head when the iris begins to bulge or pain is felt. The eye is illuminated by a Hess Hammer Lamp. The operator faces the eye in such a way as to get the best possible view. Further steps of the operation are done with the patient on the operating table either with the hand magnet or with the giant magnet. The series was done under the same conditions, magnets and by the same operators.

In 61 cases, there were 11 enucleations, or 18%. Of these, two were primary because of panophthalmitis with very large foreign bodies. In two cases, the foreign body was impaled in the sclera and could be removed with difficulty only after enucleation. In two cases, the wound of entry was scleral, near the ciliary body and had therefore led to progressive hypotony. In two further cases, there was marked initial infection with exudate into the anterior chamber and extensive hemorrhage in the vitreous which also led to the soft eyeball which it was not safe to retain. In one case, vitreous abscess was present and continued to develop after the extraction. Another had massive vitreous hemorrhages and a + Wasserman with protracted inflammation which necessitated enucleation to safeguard the other eye. And finally, one case with massive vitreous hemorrhage and increasing inflammation during eight days; three X-rays localized the F. B. on the retina or within the sclera at the posterior pole. Posterior extraction was attempted with insertion of hand magnet. There was ultimate shrinking of the eye. Enucleation showed this to be a double perforation and that operative interference should have been omitted. This type of case, of which we had several, was a difficult problem. We have found it advisable to err on the side of conservatism as we found "watchful waiting" might be rewarded by the detection of double perforation as the vitreous cleared and the inflammation subsided as is usually the case even if the F. B. should prove to be intraocular. The risk of letting an intraocular F. B. rest for a few weeks would seem to us the lesser of two evils. We are confirmed in this view by the review of our extractions of foreign bodies of several weeks to months standing even in the presence of initial infection. In this connection, one may recall Haab's series of 14 cases of intraocular foreign bodies of several years

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standing without siderosis or reduction of vision. In summarizing, one may observe that the enucleations were, with one exception, unavoidable and due to the nature or extent of the primary insult.

Siderosis was present in 8 cases, or 13%; in 7 cases extraction was successful and such vision as was present was retained or subsequently improved by extraction of cataract. In one case, the F. B. could not be extracted. Of especial interest were—one case of but one month's standing with siderotic amaurosis, size of foreign body 3 x1 x 1 mm., and another of six months' standing, F. B. on ciliary body and vision of 8/10. In the latter case, 0.8 vision was retained following extraction by the posterior route. After one year, there was gradual development of cataract which was extracted. In spite of black pupil and clear media, there was vision of but light perception. Siderotic degeneration of the retina therefore had progressed after the extraction of the offending F. B. itself.

The time elapsed since the entry of the various F. B.'s varied from one month to four years. In most of these siderotic eyeballs, convalescence was smooth, nor was there delayed healing. The same applies to subsequent cataract extractions, in which it was incidentally noted that the capsule was considerably thickened. Finally one case was seen after complete absorption of the F. B. with siderosis occurring and recent development of glaucoma. Of eight siderotic eyes, five were practically blind, due to siderosis. If one considers that the extraction offered no difficulties, it is apparent how easily this loss might have been avoided for the injured as well as for the insurance carrier if the operation had been performed at an earlier date.

Of the successful extractions, twenty-four were done by the anterior route and twenty-five by the posterior.

In the series of posterior extractions, there were four enucleations which have already been detailed and seven cases of retinal detachment, two of which became reattached with good vision. In fifteen vision was the same as —or better than—before extraction. In five other cases with almost no vision, the site of perforation was scleral and the F. B. large.

Of the anterior cases, twenty-one extractions were successful inasmuch as the vision remained the same or was improved by extraction. Two cases came to enucleation, noted above. The lens was not injured during the course of extraction in a single case. Nine iridectomies, however, were performed.

A comparison between the results of the anterior and the posterior routes is obviously not a fair one for the cases are selected for the operation—the good ones for the anterior, the bad ones for the posterior, route. At other times, when the anterior route has failed in its purpose, the onus must be borne by the posterior route. The posterior route indeed often plays the role of the stepchild; it is given the large and jagged pieces, those tied down by scar tissue or those which have torn through the ciliary body on their entrance. The posterior route may, therefore, be pardoned a host of sins but not that of retinal detachment which, in our series, totalled seven, five of which were definitely postoperative. That two of these became reattached with good vision may be regarded as undeserved and the exceptions which prove the rule. The detachments occurred some three or four months after operation and it is important to note that, in these very cases, the operative notes, dictated on the day of operation specified on each occasion—"Exceptionally smooth, no traumatism or vitreous loss." Haab, in reviewing the literature of posterior extractions, calls attention to such operative notes as the above and indicates that they have no bearing on the case. Here we entirely agree with him. Although the posterior route commands the field in certain cases, we feel that its comparative ease of performance should not broaden its scope but that the anterior route should be used whenever it is at all possible. The difficulties of the successful execution of the latter are considerably greater but, in our experiences at least, the assurance of a good end result with preservation of vision has been much greater. We regard the exact localization with the Sweet localizer by an expert in this particular field

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and a thoughtful consideration of the size and the location of the F. B., as well as intimate contact and consequent cooperation of the patient previous to the operation, as part and parcel of the operative procedure itself.

The bugbear of the anterior route appears to be the danger of the F. B. becoming stuck in the iris on its way to the pupil and the consequent necessity of an iridectomy. It is true that in our series two of the iridectomies were premeditated as the patient had undergone several attempts at extraction elsewhere with resultant impalement of the F. B. in the ciliary body. In such cases an iridectomy, followed by insertion of the tip of the hand magnet into the posterior chamber resulted in smooth extraction. Yet, the percentage of iridectomies is higher than one would wish.

In a painful, irritated eye, it is not always possible to achieve maximum mydriasis. Haab has suggested the injection of 10% cocaine with a drop of adrenalin to obtain anaesthesia in those cases where the F. B. presented behind the iris. He states in this connection that the local dilatation of the pupil, due to the cocaine, may incidentally facilitate extraction of the F. B. through the pupil. It occurred to us that a subconjunctival injection of adrenalin near the limbus might solve a number of difficulties and obviate what we regard as the main hazard of the anterior route. We have been using subconjunctival adrenalin injections to dilate the pupil, break down synechiae and in glaucoma secondary to iritis, ever since Hamburger's first publication with most gratifying results. We found that it gave much better results than the injection of atropin and that the latter can be dispensed with. One gained the impression that the stimulation of the dilator, apart from all other considerations, is much more important and useful than the paralysis of the sphincter with atropin. It occurred to us that in certain intraocular operations a mydriasis due to tonicity of the iris would be preferable to a mydriasis due to paralysis of the iris. One might say that in a "tonic mydriasis" there should be less tendency to prolapse of the iris upon opening the anterior chamber than in a "paralytic mydriasis." Certainly the anaesthesia should be improved and, if the hypotension did not interfere with the ease of operative manipulation, it, in itself, should tend to prevent prolapse of any of the contents of the bulbus. This indeed proved to be the case. In our last seven cases of intraocular steel extraction by the anterior route, we injected five minims of adrenalin subconjunctivally near the limbus with the third drop of cocaine. Within ten to fifteen minutes the pupil had maximally dilated, anemia was marked with consequent absence of hemorrhage and excellent anaesthesia. The hypotony did not disturb in the least. The guidance of the F. B. into the A. C. was much facilitated and we received the impression that the tonus of the dilator actively retracted the iris from the F. B. as the latter approached and passed the pupillary margin. Interestingly enough, the pupil contracted somewhat after the passage of the F. B. into the A. C., no doubt due to the mechanical irritation during its passage. This was not unwelcome. The extraction of the F. B. from the A. C. with a hand magnet seemed easier than with the atropinized pupil. There seemed less tendency of the iris to follow the F. B. or to prolapse and there was no need of reposition. We found no record in the literature of subconjunctival adrenalin as a preoperative measure in magnet extractions and we hope that this suggestion may prove fruitful.

Age and vascular disease will set limitations upon its employment but, in selected cases, it would seem to be a measure of promise.

Discussion opened by Dr. Joseph Goldstein, Los Angeles:

Dr. Barkan has done us a great service in emphasizing the value of the anterior route for extraction of intra-ocular F. B. and for calling our attention to the fact that a short delay in order to permit the tissues and media to subside is often to be preferred over an immediate intervention.

Their good results and experiences in which siderosis had commenced or was considerably present is also encouraging. We believe with them that extraction should be done prior to siderosis if possible.

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Their percentage of successful extractions, practically 50% by the anterior as well as 50% by the posterior route, is interesting and should encourage most of us who have leaned to the posterior route, to try out more often the anterior extraction.

The number of iridectomies by the anterior operation, to a great extent, depends upon the skill of the operator.

A most valuable contribution is added, as Dr. B. points out, by the injection of adrenalin. With a maximum "tonic mydriasis," it would appear off-hand rather unusual to have a F. B. engage in the iris. However, it was noted that seven iridectomies were necessary in twenty-one cases. I take it that a greater percentage of iridectomies were done prior to using adrenalin and this probably accounts for the high percentage.

We have had no experience with adrenalin as advocated in this paper and are much interested in the action of the iris and the results obtained thereby. Their experience would indicate that we had a valuable aid in this work.

Lately, my attention has been directed to some patients who were operated years ago by the posterior route. Recently, without any definite cause, or with just the remotest cause, something has happened and the vision was lost. This has been noted by other men. Will Dr. Barkan kindly inform us why such a condition should arise in a quiescent eye?

It is unfortunate that we are not able to follow the histories of more of these cases years after they have been operated. Some interesting data might result from such an observation.

The old German idea wherein it was taught that every F. B. should be removed or the eye enucleated, must be altered. The literature abounds in cases where eyes have given useful vision for twenty, thirty and even forty years.

It gives me great pleasure indeed to be able to lead this discussion on my own professor's excellent paper.

Dr. Kaspar Pischel, San Francisco:

A foreign body should be removed from the interior of the eye as soon as possible; every hour's delay means additional danger of infection or irritation.

How quickly changes may take place, I noticed the other day. I saw the patient with a piece of steel in the vitreous at 11 a. m.; sight normal. The ophthalmoscope revealed a small retinal hemorrhage at the point of entrance of the foreign body. Five hours later the sight was reduced to counting fingers; the retina was covered with hemorrhages.

When a patient comes with a fresh injury I have immediately a Roentgen picture taken and the foreign body localized by markers in the conjunctiva (Transactions, 1925, page 87). This can be done in a few minutes by any capable radiographer, while the Sweet localizer can only be had in larger medical centers.

For quick dilatation of the pupil I found injection of cocaine 5%, adrenalin 1/1000, atropin 1%, equal parts, 1/3 CC, very effective. Glaucosan might be even better.

Regarding the route of attack, I consider the posterior route advisable, if the foreign body is behind the iris plane. I make an incision through the sclera and introduce the small tip of my giant magnet, thus approaching the foreign body as much as possible, which is of great importance, as the drawing power decreases the square of the distance.

Dr. Jordan, San Jose, Calif.:

There are two points I wish to bring out on this subject. First, patient should not be kept by family physician, and second, if foreign body is in posterior pole radiogram should be taken on rotating axis to determine if foreign body moves with globe.

A little boy was pounding with a hammer. He came in and said a bug

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had gotten into his eye. He had a delightful family physician who very thoughtfully kept him for one week distilling argyrol into the eye. He should have been sent to an oculist at once.

A radiogram was taken using the Sweet Localizer, and the foreign body was located in the posterior pole of the eye. Then a composite radiogram was taken, i. e., two exposures were taken on the same film. The head was kept motionless, but the globe was rotated for the second exposure, showing two pictures of same foreign body. The foreign body moved with the rotation of the globe, showing it to be within the globe. The eye was enucleated after fruitless attempt at extraction.

Dr. Cameron, Tacoma:

Dr. Goldstein called attention to the fact that the eye tolerates a foreign body for considerable length of time and as we heard in our discussion the other morning, regarding Sympathetic Ophthalmia, we are not seeing the cases in these days that we did in the past. To my mind, that is due to the fact that we are watching these cases and taking care of focal infections. By watching a case carefully and clearing up the teeth and tonsils and any area of focal infection we can avoid a good deal of trouble.

I have in mind a patient who had fragments of copper in both eyes due to explosion of a dynamite cap. One eye was destroyed and subsequently enucleated. There were pieces of copper in the iris and lens of the remaining eye. The lens gradually absorbed and he regained good vision with a cataract lens. All sources of focal infection were given attention and his eye has remained quiet for the past seven years.

A boy had a small piece of steel in the posterior part of the eye imbedded in the sclera. Vision was 20/20. The steel was removed by scleral route, but the eye was lost by subsequent infection. I believe it would have been better surgery to allow the steel to remain in the sclera, meanwhile guarding the eye against any endogenous infection.

Dr. Weeks, Portland:

With regard to foreign bodies in the eye and the ultimate result of the retention of the foreign body in the eye, I would like to refer the gentlemen to the writings of the late Prof. Hirschberg on the subject. He has traced the after effects of a very large number of cases of retained foreign body in the eye. As a result of his observation he is convinced that eyes in which metal enters should be removed, unless the foreign body can be extracted.

Dr. Otto Barkan (Closing):

Answering Dr. Jordan's question in regard to deciding whether a foreign body at the posterior pole is inside or outside of the eye by taking pictures before and after rotation of the eye, I do not think that this is of much value as a foreign body just outside of the eye and within Tenon's Capsule will rotate with the eye just as if it were intraocular.

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CHOKED DISC IN INTRACRANIAL LESIONS THE MECHANICAL FACTOR IN ITS CAUSATION

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In a review of the literature Parsons[1] outlines the history of the pathogenesis of papilledema of the optic nerve as follows: Turk in 1853 was the first to draw attention to the ophthalmic findings in brain tumor, chiefly retinal hemorrhages; in 1860 von Graefe described choked disc in its relationship to intracranial diseases. His conception was that a tumor mass caused pressure directly or indirectly upon the cavernous sinus, thus producing congestion of the ophthalmic vein and central vein of the retina, leading to edema of the papilla and hemorrhages. Following von Graefe, Sesemann and Judeich questioned the theory of pressure on the cavernous sinus owing to its anatomic structure.

The next theory advanced was that of Schmidt-Rimpler, who advanced another mechanical theory, based upon Schwalbe's demonstration of the continuity of the subarachnoid space with the vaginal space of the optic nerve. He attributed the edema to increased intracranial pressure impeding the flow of lymph from the eye so that fluid is pressed out of the distended vaginal space into the lamina cribrosa. Manz and Deyl elaborated on this theory, also Parinaud and Ulrich. Their theories were not fundamentally different from that of Schmidt-Rimpler.

In 1811 Leber advanced the inflammatory theory of choked disc and was followed by other ophthalmologists who held to both the mechanical and inflammatory or a combination of the two theories. Hughlings Jackson was the first, followed by Benedickt, to attribute papilledema to a vasomotor disturbance. Chemical reaction, due to metabolic products, was advanced by Kruckmann and others. Experimental work was done by Manz, Parinaud, von Schulten, Deutschmann and others in an attempt to produce choked disc by means of increasing the intracranial pressure. Merz produced edema, with blurring of the edges in from 8 to 12 hours; others failed to corroborate his experiments, however.

From 1890 on until 1898 when Loeb published his experiments on the ligature of a frog's leg with a subsequent edema due to an acidosis, both the mechanical and inflammatory theories were advocated by various writers as the cause of papilledema. After Loeb's work in 1898 and the work of Cannon in 1901 the factor of acidosis became quite important in all theories and discussions regarding choked disc. To quote Parson's concluding paragraph:

> "In those animals in which the ocular blood supply is mainly of cerebral origin one may expect high intracranial pressure to be accompanied by slowing of the orbital and intra-ocular blood flow, with resultant malnutrition. Why the edema of the optic nerve should manifest itself almost entirely at the intra-ocular end must be due to local causes, and these are not far to seek. A very slight edema of the nerve at the lamina cribrosa will compress the capillaries, lead to greater anemia and greater edema, until finally the larger vessels are also compressed. The fact that papilledema is at first a pure edema is in favor of this view. It is only later, when the products of disintegration have accumulated and act as a chemical stimulus, that infiltration with leucocytes occurs. It would be unwise to press the hypothesis too far, but it may be of value in guiding future researches."

Our present conception of the etiology of choked disc began with the exhaustive researches of Leonard Hill on the cerebral circulation. We must first consider the anatomic conditions underlying the cerebral circulation in the damming-up, the difference between the intracranial circulation and the intraocular circulation as regards expansion of the enclosing membranes, and the effect of arterial tension upon these two structures. Second, we must consider the venous circulation and the effect of increased intracranial

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pressure upon the venous sinuses within the cranium and the indirect effect of venous stagnation in the sinuses upon the cerebrospinal fluid circulation. *The three factors that probably play the most important part are, then, arterial circulation, both intracranial and intraocular, the venous circulation, particularly intracranial and to a lesser degree the cerebrospinal fluid circulation.*

There can be no question but that anemia brought about mechanically to any part of the brain causes a chemical reaction, variously termed acidosis, reaction to metabolic changes, or under some other heading. Experimental work has been very difficult because of the fact that animals of lower order, such as rabbits, dogs and cats, which are commonly used in experimentation, have a return supply of blood from the eye through the extra-cranial route, whereas in the human practically all of the blood from the eye is returned to the venous circulation through the intracranial route[2] (Davis[3]) in 1925, was able to produce choked disc in the eyes of a dog by imbedding in various parts of the brain agar-agar in a gelatin capsule. He makes no mention of ligating the extra-cranial veins carrying the blood from the eyes of his dogs. He was able to produce choked disc at will and to relieve the choked disc by means of the classical sub-temporal decompression. Harvey Cushing[4] has contributed largely to our knowledge of the circulation of the fluids in the subarachnoid spaces and the production of papilledema experimentally.

At the present time, to quote Emory Hill[5]:

> "Our present knowledge of the cerebrospinal fluid spaces and their contents, coupled with clinical experience, points convincingly to the conclusion that choked disc is a mechanical effect of increased intracranial pressure. Beyond this general statement we can hardly go without speculating in regard to much disputed questions. For example, the importance of a dilated vaginal sheath of the optic nerves, whether this be a late or an early development in papilledema; the relative importance of pressure of fluid in the perivascular spaces about the central retinal vessels in the nerve and of pressure surrounding the nerve; the possible elaboration of chemical substances which damage the retinal tissue and blood vessel walls in the course of tumor growth, or of hydrocephalus without tumor; the exact locations at which a block of the cerebrospinal fluid channels will produce the highest degree of stasis transmitted to the optic discs; the explanation of the subsidence of papilledema when the excess of intracranial pressure has not been relieved. These and other questions deserve study, but are outside the domain of clinical ophthalmology."

My interest in the etiology of choked disc began about 1912 when I had occasion to observe a gradually developing choked disc in a person suffering from a tumor of the parietal lobe. This patient was subsequently operated on by a general surgeon with the classical decompression operation. The choking subsided for a time and then returned and was followed by complete secondary optic nerve atrophy. A large hernia cerebri developed in this case, almost as large as the man's head. He lived for two years following the decompression operation. Subsequent observations were made on a number of clinical cases, each with different symptoms. Repeated searches through the literature for theories which would explain the various phenomena failed to reveal a satisfactory explanation in these cases. For instance, all neurosurgeons have had occasion to observe large tumors in the frontal lobe with a very late production of choked disc, whereas small tumors in the temperosphenoidal or cerebellar region would produce enormous choking.

Traumatic surgery has taught us much regarding the acute development of choked disc and can furnish convincing proof that the mechanical factors alone can produce it. For instance, a patient,[6] referred from Alaska, whose head had been caught between two ore cars causing fracture of the base involving the sella and in which a double arterio-venous aneurism developed with resulting choked discs. It came on almost immediately, as the doctor in Alaska reported, and the patient presented a typical picture of a cavernous

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sinus thrombosis. Here we have a simple mechanical production of choked disc. All of us have seen similar conditions developed in a thrombosis of the cavernous sinus or in aneurisms as described above. Ligation of the internal carotid in this instance relieved the bruit on one side and the choking subsided. One would have expected with the increased work put upon the opposite internal carotid that the aneurism of that side would increase and yet it apparently remained static. In another case, seen in consultation with Dr. E. O. Jones, in which, following a skull fracture due to an automobile accident, there developed a choked disc and arterio-venous aneurism of the right side, a glaucoma developed in the eye. Subsequently the cartoid was ligated on that side and the glaucoma subsided as well as the aneurism. This would tend to corroborate the theory of Parker[7], that there is an increased intraocular pressure in all the cases of increased intracranial pressure. This has not been observed in other cases, nor have we felt justified in using a tonometer in cases where choked disc are plainly evident.

Fort Riley, during the influenza epidemic, offered an opportunity[8] to observe many cases of acute pneumonia with slight delirium of a one or two days duration. The autopsies in these cases revealed a marked stagnation of the blood in the intracranial sinuses. The cavernous sinus sometimes would stand out in marked contrast to the adjacent dura on the floor of the middle fossa. The lateral ventricles were also markedly dilated. The capillaries on the surface of the brain would show the same intense dilatation as on the surface of the optic thalami. It appeared that here we had a typical damming up of the venous return such as one would get when injecting a foreign substance under the dura. An observation was made at that time that perhaps the engorgement of the ventricles was due to a failure on the part of the longitudinal and transverse sinuses to carry away the cerebrospinal fluid as quickly as it was formed.

While still believing that the cerebrospinal fluid played an important part in the production of choked disc, observation was made on a series of cases of acute pneumonia and in each instance the eye grounds showed a beginning papilledema. The changes varied from a filling of the physiological cup to the typical upper and lower blurring of the disc margin and finally to a true papilledema. In those cases that we were permitted to observe over a longer period of time, with the recedence of the delirium came a recedence of the edema of the retina. In this series of cases we had to deal with an acute inflammatory condition which brought about a stagnation of blood within the cranium. A generalized edema followed and in each instance, an increased intracranial pressure.

I then became a convert to the purely mechanical theory of choked disc. Subsequent work in the field of brain surgery, with observation of a number of cases, has convinced me that it is the greatest factor with which we have to deal, and that this mechanical factor may often be brought about by the presence of a foreign material, such as a tumor, which may cause, first, a local edema of the brain with later a general edema of the brain, which in turn, acting mechanically on the venous sinuses, causes the damming-up of the fluid.

A series of illustrations will show how a lesion may be so situated as to produce a unilateral choking, a bilateral choking, early or late choking, as the case might be. We have used in these cuts the diagram found in "Anatomy of the Brain and Spinal Cord" by Whitaker and have placed the lesion in different parts so as to illustrate the point.

Figure 1 illustrates the anastomosis between the vessels of the choroid and optic nerve after Leber (P. 950, Fig. 676). It shows how the slightest amount of pressure can cause a damming-up of circulation not only of the vessels of the nerve itself, but of the choroid as well.

Figure 2 illustrates a lesion in the right cavernous sinus. Such a lesion, as for instance, an aneurism due to skull injury or a cavernous sinus thrombosis due to infection, both of which produce a uni-lateral choked disc very early. The case cited of Dr. Jones and many other similar instances could

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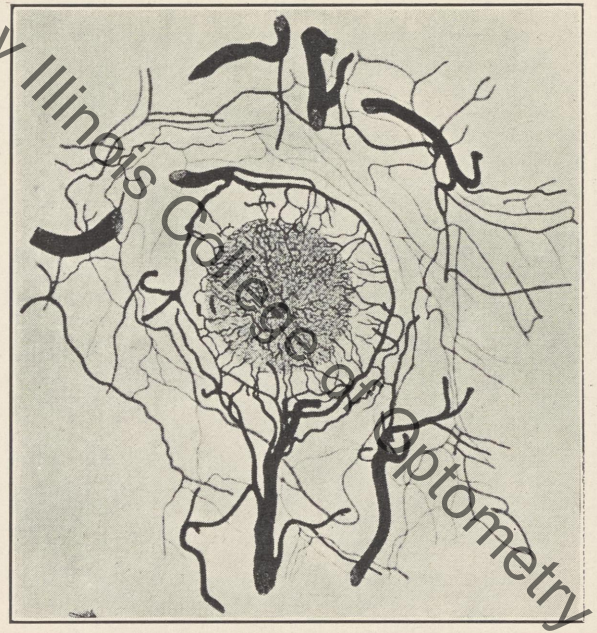

Fig. 1. Showing anastomosis of vessels of disc and choroid (from Parsons after Leber.)

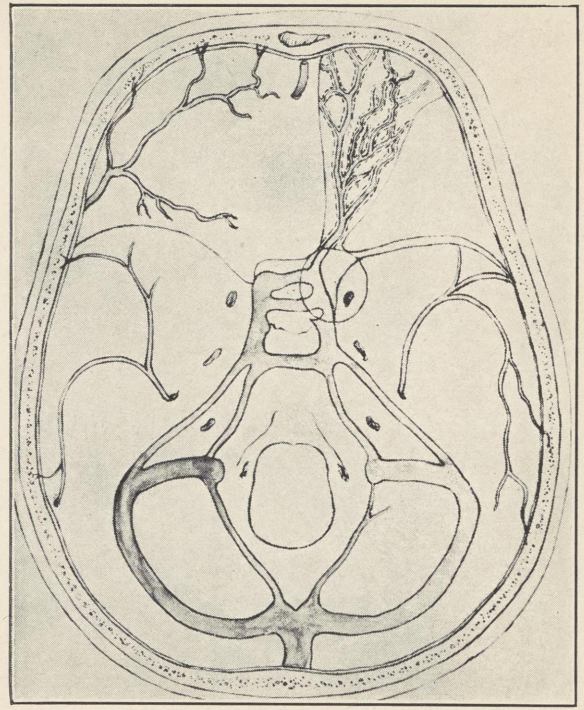

Fig. 2. Lesion of cavernous sinus, causing sudden and early choked disc (outline from Whitaker's Anatomy of Brain and Spinal Cord).

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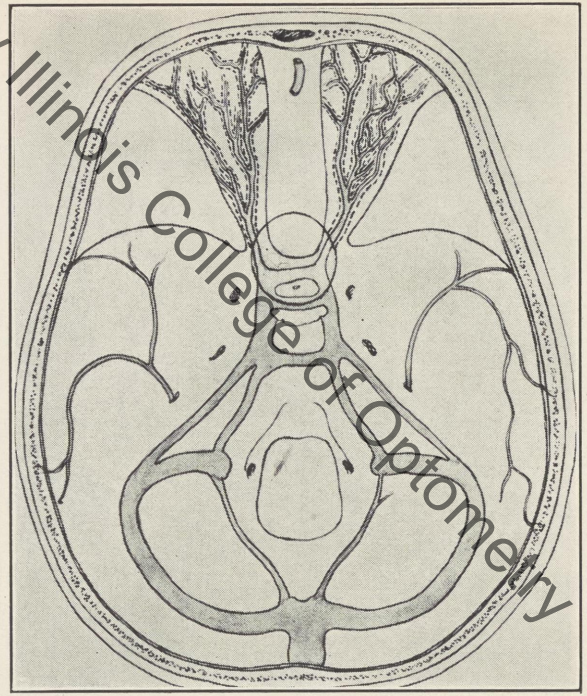

Fig. 3. Lesion of pituitary gland or aneurism of circle of Willis. Choking a terminal sign.

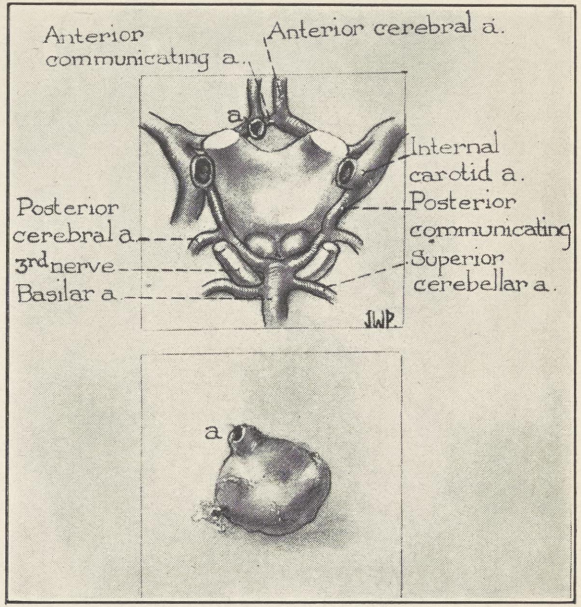

Fig. 4. Aneurism showing marked distention and flattening of optic chiasm. Same as fig. 5.

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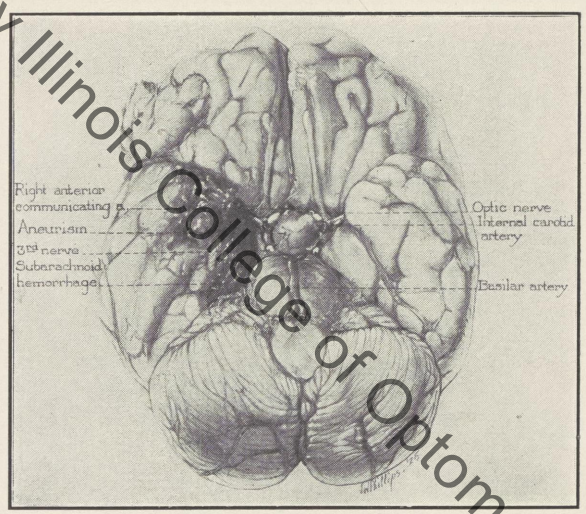

Fig 4a. Aneurism of circle of Willis with no choking until after large hemorrhage into cerebral hemisphere of right side.

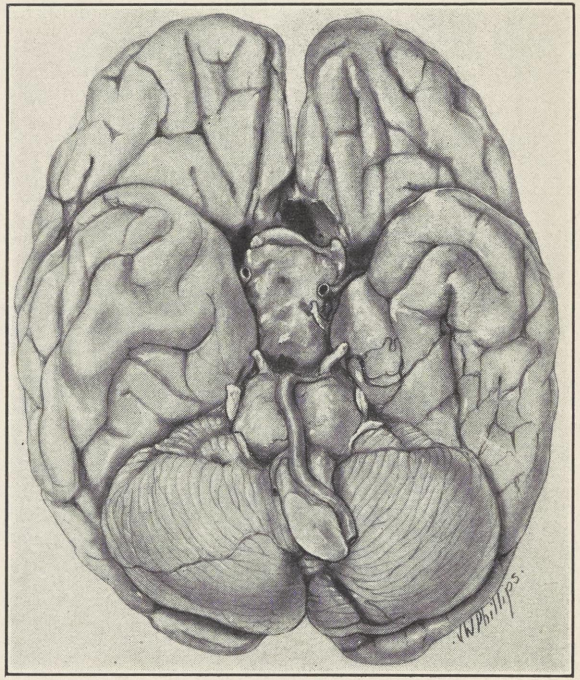

Fig. 5. Aneurism of circle of Willis, eroding into sphenoid sinus. Choked disc on right side late in the course of the illness.

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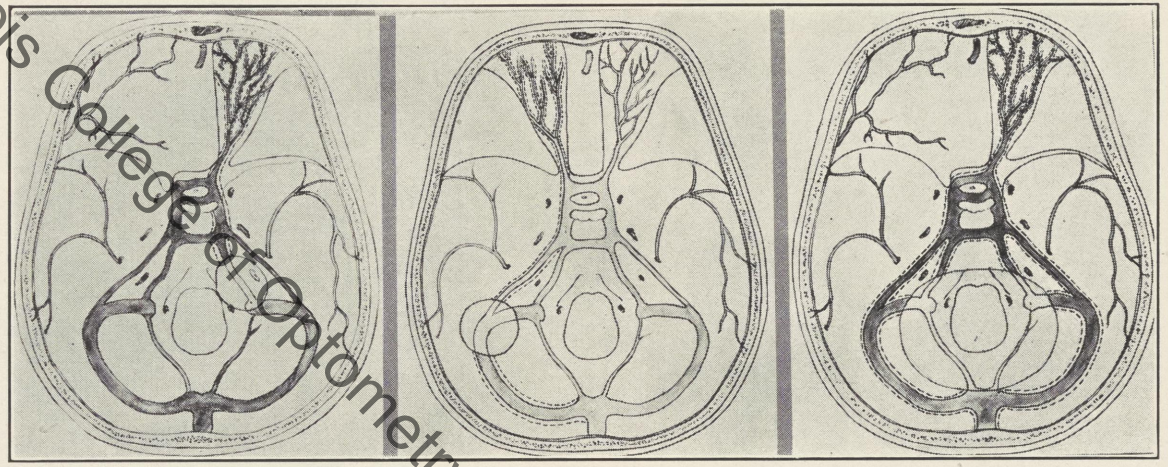

Fig. 6. Cerebellopontile angle lesion.

Fig. 7. Lateral sinus thrombosis.

Fig. 8. Lesion in cerebellum.

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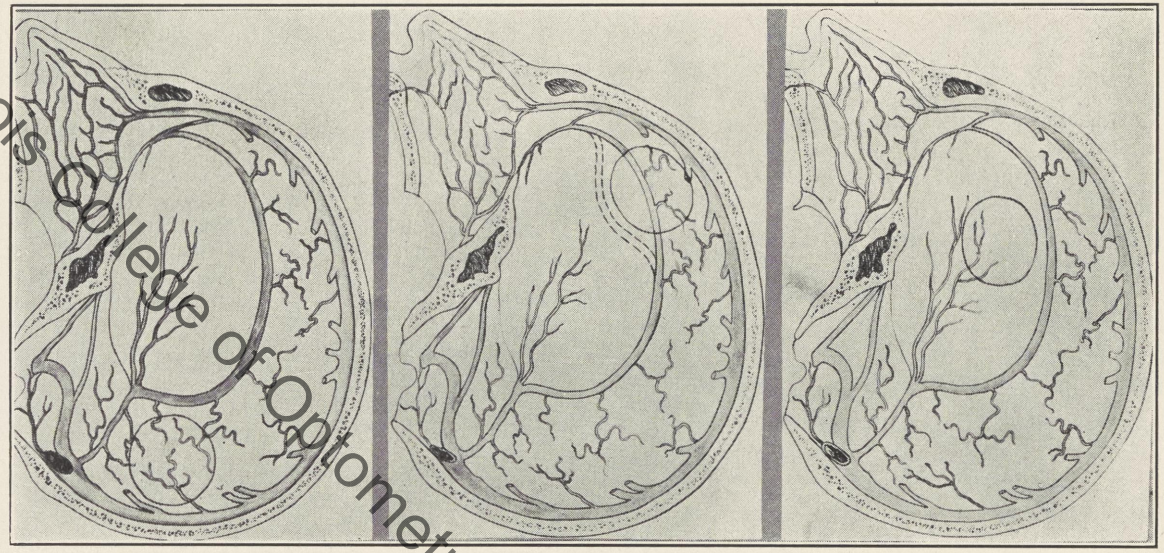

Fig. 9. Lesion of occipital area.

Fig. 10. Lesion of frontal area.

Fig. 11. Lesion situated in central nuclei area.

be used to illustrate this point. Why does not a lesion of the optic nerve such as, for instance, a neuroma of the sheath produce a choked disc rather than optic atrophy? First of all, as the tumor grows in the orbit the bony wall gives away or the eyeball bulges forward and while the veins are stretched, they return through the spheno-palatine fissure and there is not a direct pressure on the cavernous sinus or the veins themselves.

Figure 3 shows a lesion in the mid-line which might cause a double choking. Such a lesion occurred in the first case reported, that of a double aneurism of the cavernous sinus. A large tumor of the pituitary, or an aneurism of the circle of Willis, pressing lateralward against the chiasm, would account for local pressure at this point which may cause a double choked disc.

Two aneurisms, the one small, the size of a large hickory nut, situated immediately over the optic chiasm and pressing against the chiasm, as shown in the drawing (Figures 4 and 4a), so that it was flattened and thinned in the center, did not cause a choked disc. The second (Figure 5), an aneurism which had eroded the bone in the body of the sphenoid sinus did not cause choked disc until just before death. At that time the symptoms developed of a 3rd nerve paralysis, 5th nerve initial irritation, later paralysis and finally a 6th nerve paralysis. It was upon this progressing chain of symptoms that the diagnosis was made just prior to death.

In Figure 6 a lesion involving the angle of the cerebellum, pons and medulla again causes, first, a uni-lateral choked disc and later a bi-lateral choked disc. Figure 7 is similar to Figure 6 and shows what occurs in a thrombosis of the transverse sinus in mastoid disease. In fact, in these cases a choking always develops first on the side of the lesion. Wanamaker has reported a series of cases of this character and others have made similar observations. The Queckenstedt test probably is more accurate than the eye examination and we have used it for several years to determine the presence or absence of a transverse sinus thrombosis.

Figure 8 shows the typical mechanism of a tumor situated in the posterior fossa. A cerebellar cyst, abscess or tumor at the angle produces a double choked disc. This is brought about by pressure against the transverse sinuses as well as damming up the blood from the petrosals. The venous return from the superior longitudinal sinus as it reaches the torcular divides and leaves the posterior fossa through the two transverse sinuses. Just as it enters the floor both sinuses receive the blood from the inferior petrosals, which carry the bulk of the blood from the cavernous sinuses.

The first effect, then, of a tumor in the posterior fossa is to dam up the blood from the petrosals, which in itself will cause a choked disc rather early and, if the tumor is situated on one side, usually the choking begins in the eye of the same side. The second change is that of lateral pressure against the walls of the transverse sinuses. This dams up the blood from the superior longitudinal sinus, which in turn causes a stagnation of the cerebrospinal fluid over the cortex of the brain. It likewise obstructs the aqueduct of Sylvius, so that in addition to the choking we have a uniform distension of the lateral ventricles.

The intracranial pressure in these posterior fossa cases is always much higher than in tumors situated elsewhere, and likewise in decompression operations in these cases one is impressed by the copious flow of cerebro-spinal fluid under intense pressure which results immediately after puncture of the dura. These mechanical factors thus explain the two results, namely, choked disc and increase of cerebrospinal fluid pressure.

In the three subsequent Figures, 9, 10, and 11, the location of the tumor is seen to be where it is almost impossible to cause a damming-up of the petrosal sinuses, until such a time as the general increase of intracranial pressure causes a damming-up of the venous sinuses, and thus one can readily see why a large tumor of the occipital lobe will show practically no choking of the disc until the terminal stage. A frontal lobe tumor, Figure 12, may also reach an enormous size before choking occurs.

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In Figure 10 the tumor is situated either in the optic thalami, the corpus callosum or in the third ventricle. Tumors of the optic thalami never show early choked disc (Figure 13). Some of these tumors are of enormous size and extend well into the substance of the hemisphere in which the lesion occurred. Cysts and tumors of the pituitary, Figures 14 and 15, likewise can be of very large size before choking occurs, because of the fact that they extend into the substance to either side, but always above the cavernous sinus and, therefore, do not cause a retardation of circulation until the terminal stage.

Conclusions:

(1) Choked disc or papilledema is always either the direct or indirect result of a damming-up of the return venous blood from the eyeball.

(2) The time of its appearance depends upon the location of the tumor and occurs in the following sequence: (a) Cavernous Sinus, (b) Petrosal Sinuses, (c) Lateral or Transverse Sinuses, (d) Occipital Fossa, (e) Parietal Fossa, (f) Frontal Fossa, (g) Central Nuclei.

(3) Direct pressure, as against the petrosal or cavernous sinuses, causes early choked disc while indirect pressure, as in edema of the brain, or dilated lateral and third ventricles, or both, causes late choked disc.

(4) The cerebrospinal fluid plays but an indirect part in the causation of choked disc, and then only by increasing the general intracranial pressure and not by direct extension into the disc from the vaginal sheath of the optic nerve.

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Discussion opened by Dr. Walter B. Lancaster, Boston:

I want to thank Dr. Swift for the opportunity of discussing his paper. I think it is an important paper and that it should have a much better discussion than I can give it. Dr. Swift sent me a copy of his paper, but I live so far away that I did not receive it in time to look up the literature and the important experimental work that has been done on the subject.

The question which was so thoroughly threshed out a few years ago was whether Choked Disc was of infectious, toxic or inflammatory origin on the one hand, or purely mechanical on the other. This was definitely settled in favor of the mechanical theory. When there is a swelling of the optic disc of toxic inflammatory origin, we have a true optic neurtis, not a papilledema or choked disc. Now Dr. Swift, accepting of course the mechanical theory, goes on to claim that the papilledema is not due to the cerebrospinal fluid being dammed back in the optic nerve, and so causing edema, but is solely due to the blood stream in the veins of the optic nerve being dammed back and so causing edema from venous stasis.

I believe that Dr. Swift goes too far, that he underrates the importance of the cerebrospinal fluid; but I admit that the vascular factor is important, and that we are deeply indebted to Dr. Swift for calling our attention to it.

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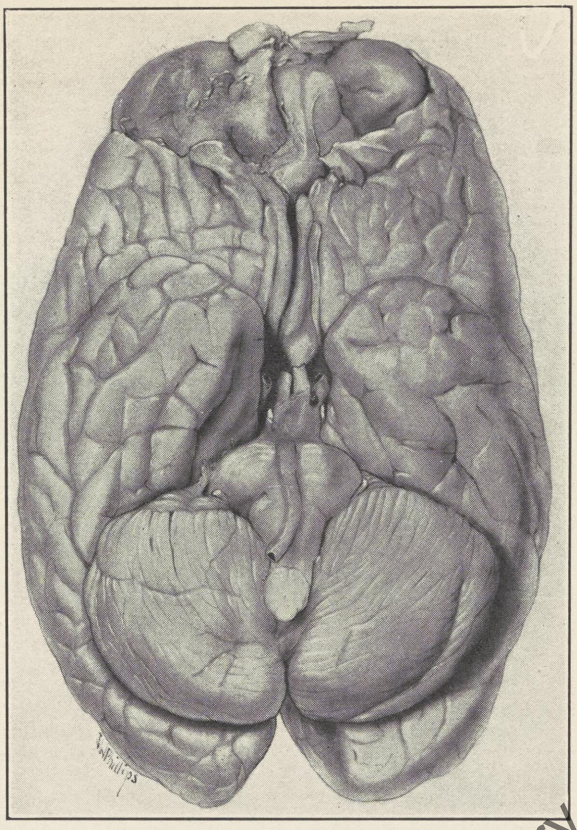

Fig. 12. A large frontal lobe tumor with choked discs as a terminal sign. (Metastatic sarcoma.)

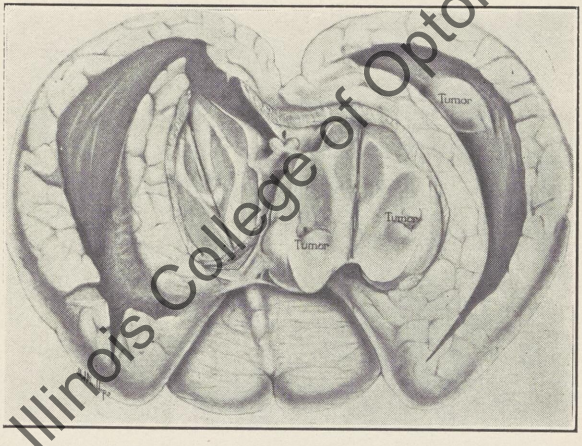

Fig. 13. A tumor (glioma) of the optic thalamus with no neurologic findings or choked disc. Patient died of sudden hemorrhage.

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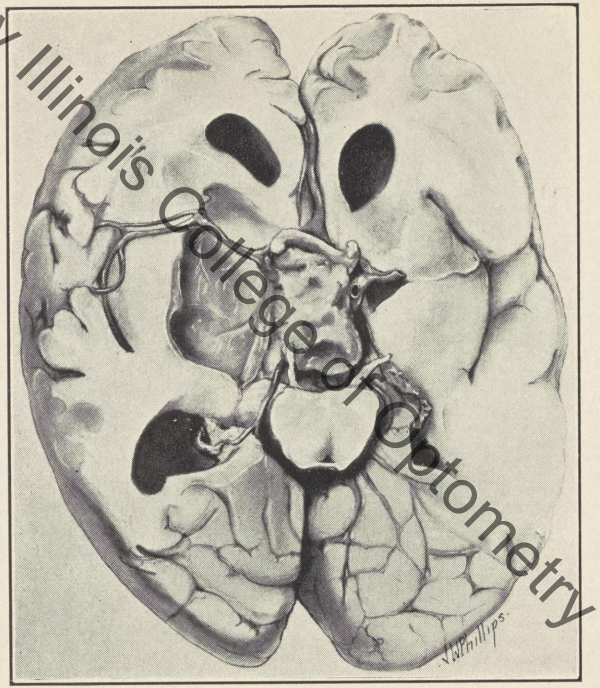

Fig. 14. Duration two years after first symptoms of a cystic adenoma of the pituitary with extension into the left temporal lobe with no choking until the last week of life.

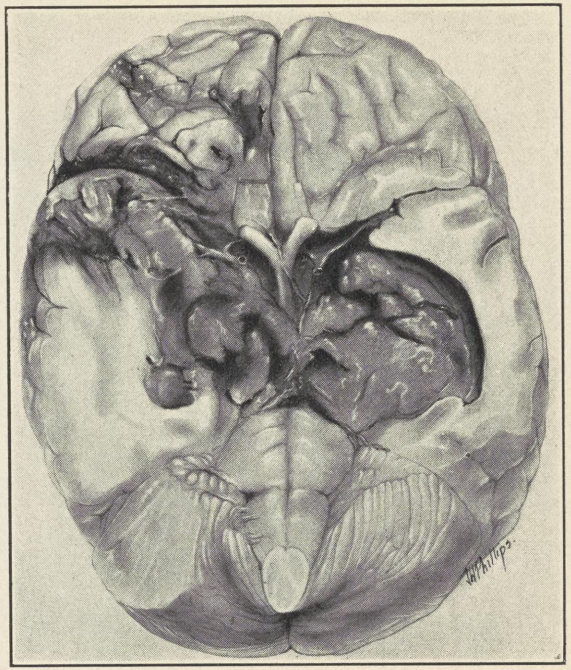

Fig. 15. A tumor of the pituitary extending well into each hemisphere and having a "terminal" choked disc.

First, I will show a couple of slides which illustrate the action of the cerebrospinal fluid under increased pressure. This slide shows the optic nerve in longitudinal section and the adjoining tissues. You see that the sheath of the optic nerve is so overfilled and distended by the cerebrospinal fluid that the space (sub arachnoid) between the nerve and its outer sheath (dura) is actually wider than the width of the nerve itself, more than ten times as wide as it is in the normal condition. If the back pressure is so great in the sheath of the optic nerve as to produce this distension, it clearly must hinder the normal outflow of fluid from the eyeball via the optic nerve, and may even reverse the flow to some extent. Incidentally this must have some effect on the venous circulation in the optic disc, and doubtless that is a contributory factor in the picture of typical choked disc with its distended tortuous veins as well as edematous swollen nerve head. Somehow it seems to me a more natural sequence—increased pressure of cerebrospinal fluid, backing up into the optic sheath, obstructing the flow of fluid from the eye via the optic nerve, then by the edema obstructing the blood flow in the veins and so completing the picture, edema and swelling of the nerve head plus venous engorgement and hemorrhages. A less natural and easy sequence is venous obstruction and consequent engorgement, followed by edema and swelling of the disc. In mild cases we get swelling and edema, with little venous engorgement, which becomes conspicuous in well advanced stages. In mild cases we do not get venous engorgement without swelling, and later see the edema and swelling added in the advanced stages.

The second point which I wish to mention has to do with some experimental work at Camp Devens. Dr. Swift says that we should be prepared to tell the physician or the surgeon a little more about what is going on than we do. I had that viewpoint when training some ophthalmologists for the army. When they got over in France and had the care of head injuries, it would be important for them to be able to say when the cranium should be operated. In order to impress this upon them, we took some dogs and trephined the skull. Then by packing sponge or gauze firmly into the cranial cavity, we produced increased pressure. We were also able to show how the swelling of the optic nerve was greater in the eye with the lower intra-ocular pressure by trephining one eye, following Parker's experiments. That is, we could produce a papilledema by increasing the intracranial pressure, and we could make the swelling greater in either eye at will by simply reducing the intra-ocular pressure in that eye by trephining the sclera. That was an object lesson to teach the men that swelling of the optic nerve showed increased intracranial pressure, that the greater swelling of one optic nerve did not depend on which side of the brain was injured, but only on which eye had the lower intra-ocular pressure, thus permitting the cerebrospinal fluid to be backed up more easily into that eye than into the other eye where the intraocular pressure was higher. Finally, the removal of the packing was followed by subsidence of the papilledema.

Another thing that has occurred to me as significant in connection with Dr. Swift's thesis that the spinal fluid is of minor importance, is the way you can reduce the pressure in the cranium by administering magnesium sulphate. This and other salts will reduce the pressure in the cranium in a very marked manner, but it is only transient. If it acts on the venous circulation, it is only indirectly through reducing the volume of fluid in the brain and especially the ventricles. The papilledema which occurs in nephritis is more easily explained as due to edema of the brain than as due to venous obstruction.

The distribution of the veins in the orbit in man is a very important matter, according to Dr. Swift's thesis. Without looking it up, I hesitate to speak too positively, but I am strongly of the opinion that besides the outlet into the cavernous sinus, there is pretty free egress for blood from the orbit forward via the facial veins, and laterally and downward via the pterygoid.

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Lastly, it must be admitted that the theory of venous obstruction helps out in many cases. There are still numerous cases where, according to our theory, and this is true whatever theory we hold, we should expect papilledema and do not find it. There are other cases where we get papilledema, and should not expect it.

Dr. Allen Greenwood, Boston:

I think we owe a great debt of gratitude to Dr. Swift, and while I do not wholly agree, I do agree that he probably has found one element and another element being the one Dr. Lancaster suggested and which was so well shown by the experiments of Dr. Walter Parker.

I had the privilege of working in the British Army during the second summer of the war in Flanders, as Ophthalmic Consultant for a group of hospitals, and the Consulting Surgeon of Area issued an order that before brain surgery was to be resorted to in our group of hospitals, the fundi should be examined by Col. Greenwood.

I also had the privilege of confirming with some of my patients in Flanders, the work of Holmes Spicer and localizing injuries to the post occipital lobes; and that brings me to a case I saw two months ago in Boston, in consultation, where a man without symptoms other than a blurred vision showed the loss of quadrant in the right upper fields. He had no choking of the disc and did not have up to the time of death. In a week's time the loss in the field of vision increased and in three weeks time the field of vision showed a complete right homonymous hemianopsia. Operation revealed quite a large tumor in the posterior occipital lobe of the brain which evidently had started below the calcarine fissure and worked up.

Two interesting features were that the brain, as revealed by operation, was under tremendous intercranial pressure and yet choked disc had not occurred. I mention this case because Dr. Swift said he had only seen one case of hemianopsia in brain tumors. The man never had a headache up to the time of death.

Dr. Weeks, Portland:

I wish to call attention to one or two points. One is that disturbance in arterio-venous aneurism of the cavernous sinus, in the fundus of the eye is not a typical choked disc. At least it has not been so in the four cases that I have observed. In all of those cases the appearance was one of enormous enlargement of veins, with exudation and hemorrhage from the veins and some oedema of the tissues of the disc.

Another point is the effect of decompression operations on choked disc. In almost all cases the choked disc subsides and does not recur until intracranial pressure increases.

In cases of tumor that develop in the pontine angle, choked disc occurs very early. In tumor of the cerebellum choked disc occurs in very nearly 100% of the cases, while in tumor of the frontal lobes the percentage is very low.

Dr. Patton, Omaha:

I wish to congratulate Dr. Swift on his excellent presentation of this subject and the very timely topic selected. Although intra-cranial pressure alone, as Dr. Greenwood and Dr. Weeks have brought out, may not in all cases be sufficient to cause diagnostic changes in the eyegrounds or visual fields, yet when we do have a choked disc in the presence of suspicious general signs, we should at least consider the possibility of increased intra-cranial pressure and the pathological lesions that are usually responsible for such a condition.

I am indebted to Dr. Swift for this valuable discussion.

Dr. Swift (Concluding):

I wish to thank the men who have been so kind as to discuss my paper. A brilliant piece of work was done by Dr. Harvey Cushing in which

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he ingeniously devised a glass window insert in the skull of a dog and then used balloons and other materials for causing an increase in pressure. He was able to demonstrate the fact that passive congestion first develops in the venous system and then a stasis of the blood or retardation of the flow. These results are now universally accepted. The last work that I have knowledge of was that done by Loyal Davis of Chicago, who was able to use agar in capsules inserted in the brain tissue for purposes of producing a substance which would displace rapidly the cerebrospinal fluid within the cranial cavity and thus produce choked disc. He was able to relieve the choking by proper decompression. In other words, he demonstrated that when one reaches a certain intracranial pressure after having displaced the compensating cerebrospinal fluid, one is able to then observe a development of choked disc and to relieve the choking by a decompression which again relieves the pressure over certain areas. This must be low down in the sub-temporal region, or, in other words, release the support to the cavernous sinus so that the blood from the eyes can again come through the cavernous sinus and escape at the jugular bulb. Secondly, by doing a sub-occipital decompression which in turn allows the fluid to escape from the jugular bulb, thus relieving the stasis in the transverse sinuses, the longitudinal sinus, and permit a release of pressure. It is my opinion that a sub-temporal decompression acts directly upon the venous vessels in the lower plane, which cause a direct retardation of the blood from the eyeballs and that a sub-occipital decompression acts indirectly in the same manner as a general intracranial pressure acts indirectly to produce a choked disc.

The points that I wish to emphasize is that choked disc is produced in a direct manner by a partial or complete occlusion of the ophthalmic vein, cavernous sinus, petrosal sinuses, or transverse sinuses at the bulb; secondly, by a general pressure within the cranium. The latter must be sufficiently high to produce a general edema of the brain and thus produce a stasis of all of the venous channels within the cranium. Third, that the onset of choked disc is earliest in those lesions situated in the following sequence: cavernous sinus, petrosal sinuses, the angle of the transverse sinus and the jugular bulb, the cerebellar fossa, occipital, frontals, and in the region of the central nuclei.

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FUNDUS CHANGES IN CARDIOVASCULAR-RENAL DISEASE

WITH SLIDE AND SCREEN PICTURES

WALTER B. LANCASTER, M.D., Boston, Mass.

Let me express my deep appreciation of the honor your president has conferred upon me in inviting me to speak to you on the subject of fundus lesions. It is evident that he appreciates the importance of reiterating old truths, of presenting again and again, perhaps from different angles, the basic principles on which our daily practice is founded. Some investigation sheds new light on some field of ophthalmology, but the light is at first seen by only a few and heeded by still fewer. Attention must be called to it again and again. As I have made no contribution whatever to our knowledge and understanding of fundus lesions, it is evident that your president wishes me to perform the very important function of selecting some of the facts and theories most in need of being emphasized and presenting them to you from my point of view.

I propose to limit the discussion to the retinal lesions associated with arteriosclerosis and nephritis, and their significance. Of the many recent articles that have appeared on this subject, I commend as most illuminating and free from erroneous observations and conclusions the writings of Foster Moore and of the workers at the Mayo Clinic, especially Benedict and Wagener. If you will read some of them you will see that I have drawn freely from them in the preparation of this paper.

The illustrations are from Foster Moore, Medical Ophthalmology, from Dr. George Derby's collection, from Dr. Arthur J. Bedell's remarkable photographs of the fundus, from slides by Dr. F. J. Verhoeff, and from some of and German text books. My obligations to them are very great and are gratefully acknowledged, for the lantern slides are the main part of the presentation.*

FUNDUS CHANGES IN ARTERIO-SCLEROSIS AND NEPHRITIS AND THEIR SIGNIFICANCE

Arteriosclerosis can often be detected in the retina before there is evidence of it in other parts of the body, not because it attacks the retina first, but because with the ophthalmoscope we can study these vessels far better than is possible anywhere else.

There is no better classification of the changes seen in retinal arterio-sclerosis than that of Foster Moore.†

(a) The phenomena seen at arterio-venous crossings.
(b) Irregularity of lumen of the arteries.
(c) Copper wire arteries.
(d) Retinal hemorrhages.
(e) Tortuosity of arteries.

Histologically the changes in the vessel walls consist in marked thickening of the various coats, especially the media and the intima. In the central artery the proliferation of the intima is most in evidence (Coats); in the tributary branches the thickening of the media predominates.

The normal vessel walls are invisible, because transparent. When they are sclerosed and thickened they become visible, and in the early stages it is easiest to see this where an artery crosses a vein. The artery conceals the vein not only (as under normal condition) for the width of the blood stream, but for the width of the vessel walls besides, because they are now

* You will find excellent illustrations of the fundus changes in Arteriosclerosis and retinitis in de Schweinitz text book (copied also in Fuchs by Duane) also in Oeller's Atlas and other well known books.

† Medical Ophthalmology, 2nd edition, page 57.

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sufficiently opaque. Ordinarily when an artery crosses a vein the vein is visible right up to the red blood stream, the walls not being seen. In sclerosis the underlying vein is obscured not only for the width of the opaque blood stream, but for a distance each side equal to the thickened arterial wall.

Foster Moore fails to emphasize another evidence and one of the most easily recognized—indentation of the vein by the artery at its crossing.

Another evidence of sclerosis is the rectangular crossing of a vein under or over an artery when they originally met at an acute angle. As a result of the thickening and increased rigidity of the artery, the course of the vein is changed so that instead of a long oblique course across the artery it takes the shortest course, which is square across. This indicates a well advanced stage of sclerosis. Still more advanced, and in fact rather exceptional, is the banking of the blood in the distal portion of the vein, due to damming by the pressure of the artery where it crosses the vein.*

(b) Irregularity of lumen. The incidence of the changes in the vessel walls is not uniform in distribution. Where the thickening encroaches on the lumen the column of blood is narrower. Proliferation of the intima is particularly responsible for reduction in the lumen, as is seen in cases of obstruction of the retinal vessels.

(c) Copper wire artery is an exaggerated brightness and modified color of the normal reflex streak seen on the retinal arteries and due to the thickened media. It is not easy to be sure that the reflex is more than normally bright, so that this is not a sign to be relied upon.

(d) Hemorrhages in the retina take their form from their site. If located in the superficial nerve fiber layer where all except the finest branches of the vessels are situated, the blood spreads between the nerve fibers—flame-shaped, striate, radiate hemorrhages. If the bleeding takes place in the deeper layers of the retina the shape of the hemorrhage is approximately round, since the anatomical structure permits the blood to spread in all directions with equal facility.

As a general rule, to which there are very many exceptions, the hemorrhages due to vascular disease (arteriosclerosis with hypertension) take the flame-shaped form because they are situated in the superficial layer. They are most numerous also in the posterior part of the eye when the retinal vessels are larger. On the other hand, the hemorrhages which are of toxic origin, such as those occurring in purpura and other blood diseases and in diabetes and nephritis, are typically the small, round, deeper-seated ones. Hemorrhages in diabetes or nephritis of vascular, not toxic, origin, would, according to this classification, be flame-shaped, because they are in the nerve fiber layer.

These small retinal hemorrhages are absorbed in a matter of a few months, but new ones are being formed so that the picture is not much changed.

(e) When the arterial wall undergoes hypertrophy it enlarges in all directions. It becomes thicker, but also considerably longer. The increase in length produces the tortuosity. The variations in tortuosity in normal vessels in the retina is so great that this is not an important aid in diagnosis.

To these five diagnostic points emphasized by Moore may be added:

(f) The silver wire arteries. Here the periarteritis has resulted in such thickening and opacity of the vessel wall that the blood stream is hidden beneath the white wall of the artery. Instead of having a red color the artery looks white. Usually this is not uniformly distributed, but shows in patches on one or a few branches. It is a mark of advanced sclerosis. It is also called "pipe-stem sheathing." It does not obstruct the lumen. It is not uncommon to find the primary branches of the central artery when they lie on the optic disc showing visible walls of a grayish color which become transparent and so invisible a little farther on. Some visibility of the vessel walls on

(* These points illustrated by slides. Good examples may be seen in Moore l. c. and de Schweinitz, copied in Fuchs.)

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the disc may occur without arteriosclerosis so that this appearance is not significant.

(g) Cork-screw tortuosity of fine terminal branches of the retinal arteries, especially around the macula, is emphasized by de Schweinitz as is also the hyperaemia of the disc which he calls "brick-red" appearance. These are not regarded as important signs by Moore, because they are rather infrequent and also indefinite.

If we take the early changes in the vessel walls as the first stage of arteriosclerosis the presence of hemorrhages is the next and that is followed later on by the appearance of tiny white spots or dots. This is ARTERIO-SCLEROTIC RETINITIS.

The white spots are small, less than the thickness of a primary retinal vein in diameter, usually not numerous, but later they may become numerous and confluent, forming good sized patches. They are frequently limited to one eye for a long time, whereas the white spots or patches of renal origin are rarely limited to one eye for more than a short time in the earliest stages.

Histologically, these white spots seem to be composed of hyaline material and to be situated deep in the retina in the outer molecular layer. They are not composed of fatty material, as are most of the spots of renal retinitis. That is, in arteriosclerotic retinitis the material undergoes a hyaline degeneration and in renal retinitis it undergoes a fatty degeneration as well. This classification is not a rigid one. The masses of fibrin in renal retinitis may undergo hyaline degeneration in some cases. Apparently it is usually in the deep layer that the hyaline material is found, but fatty cells may be found there, too, so that a definite classification is impossible until we know more about the toxic substances which cause the retinitis. In the case of arteriosclerotic retinitis it seems probable that the obstruction to the blood supply (ischaemia) is a causative factor in the production of the white spots. In renal retinitis toxic agents are more in action. The two are so often interacting and combined that here again the classification is given as suggestive, not rigid.

In advanced sclerosis the lumen may be so encroached upon that the blood supply beyond is precarious. Any slight spasm at a point where the lumen is already small may so reduce the blood supply beyond as to produce scotoma even in spite of a high blood pressure in the brachial artery. For, as Foster Moore has shown, the intra-arterial pressure in the retinal arteries may be quite low when the general arterial tension is high. Again, any cause reducing the general blood pressure may so far lower the pressure in the obstructed retinal arteries as to produce sufficient ischaemia for symptoms to appear. This may be transient or last some time. In patients with arteriosclerosis it is a rather common symptom. They complain of a blur coming over the page so that they have to stop reading. After a short rest the sight may recover. Sometimes careful questioning will bring out conditions, such as fatigue, calculated to favor the transient ischaemia. Sometimes the blur may be due to ischaemia in the brain, not in the retina.

As sclerosis of the retinal vessels advances the terminal complication is such obstruction to the flow as to cause complete occlusion.

Obstruction in the retinal veins is due to the disease of the vessel wall (phlebitis) often aided by pressure where a sclerosed artery crosses the vein. It is much rarer for obstruction to be due to thrombosis, though this may occur, especially in young patients, without general vascular disease, but with some focus of infection producing a thrombus of bacterial origin.

SIGNIFICANCE OF ARTERIO-SCLEROSIS

Having made a diagnosis of arteriosclerosis of the retinal arteries, what shall we do about it?

(a) The patient should usually be referred to his family doctor or internist, to whom a report should be made.

This raises two practical problems:

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(1) How to refer the patient without exciting undue alarm and yet secure co-operation on the part of the patient.

(2) How to report to the family doctor. Some doctors resent being told anything more than the facts—they want no advice from you.

You must use tact and judgment. It all depends on the man and on your personal relations to him.

If you can't send the patient to the internist without filling the patient with anxiety and alarm it would be better in many cases to do nothing "in the good old way." You can adopt some such attitude as this: "Some of the symptoms which you have are due to the eyes and will be helped by suitable glasses; some of your symptoms are not due to the eyes, but to certain general conditions of which signs can be seen in the eyes (we are often consulted for some of the symptoms of cerebral arteriosclerosis, such as headache, dizziness of various types and degrees, loss of memory). What I see in the eyes are indications of wear. There is no inflammation. You see by the letters you have read on the chart that your sight is first rate for your age. Now just as an automobile needs overhauling, or at least lubrication and adjustment from time to time, so does the human body. You have heard that 'no Rolls Royce ever wore out.' I suspect this is not because of its superquality. The fact is that it costs so much that owners take care of it! They have it inspected from time to time. They observe reasonable care in the way they use it. So you ought to have periodical inspections, as ever so many sensible business men and women do. You may find adjustments are needed. These may not be such adjustments as you are wont to think of as being made or advised by a doctor—not operations or drugs or manipulations. They are adjustments of your habits, modes of life, course of your thoughts, as well as diet, exercise, work, pleasures."

It may not be wise to go too fully into such an explanation. All depends on the individual and on your relations to him. One thing you can do without danger of offending the family doctor at the start is to tell the patient to report to you from time to time, making the interval fit the case.

If, after a suitable time, you find that the family doctor or internist has simply told the patient, "You are in fine condition; your heart is perfect, your kidneys are perfect, your blood pressure is a little high, perhaps, but I wish I were as sound as you are."—if you find the doctor has not given the patient any advice and you think he needs it, you can yourself give some advice.

Briefly the significance of arteriosclerosis of the retinal vessels is, 1st, that *the arteriosclerosis is not limited to the retina*. Of the other parts of the body which may be affected only three are of much importance. They are the kidneys, the heart, and the brain.

2nd—Arteriorsclerosis of the retinal vessels shows that the *causative factors of arteriosclerosis are at work*. Of the causes of arteriosclerosis hypertension and chronic nephritis, we know much or little according to your point of view. Of the actual toxic substances we know hardly anything. But we know much of great practical value about the etiology.

Among the important considerations are the following: Heredity; infections, including syphilis; overwork, mental as well as physical; discordant social life; over-anxious temperament; excess in eating and drinking; age (the earlier the onset the more malignant, like diabetes)—in short, *strain* of various kinds.

As to treatment, the application of sound common sense to readjusting the patient's life. Rational advice given early is very effective if followed. Adapt the patient's work to his capacity to carry on without strain. Make readjustments in life, social and business, slight or drastic.

If you have faith in the value and importance of this sort of treatment and realize the certainty with which the patient will go from bad to worse if he continues his faulty mode of life, you ought to be able to communicate some of your faith to the patient, because faith, like fear, is very contagious. Coax, persuade, cajole, but resort to alarming the patient with great care,

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for it is possible to do great harm by starting anxiety in a patient easily made overanxious.

I know of one wise and experienced internist who will not take on such cases unless they agree to put themselves under his absolute control for a year. He feels that it is not possible in less time to change the habits and get the new ones sufficiently established to make it safe to trust the patient to himself. I cite this to emphasize the fact that telling the patient once or twice about these readjustments is by no means doing your duty by the case. To say, "You'd better take a vacation," or "You need to cut down on your work," "Don't eat so much," "Take regular but not excessive exercise," etc., is only the beginning. Some one must see to it that the advice is followed and followed intelligently. See the patient often enough to steer him and keep him on the right course. He must be given a course on what I call *the art of living*. Some patients will be very grateful for such guidance and well they may be, for it is calculated to increase both the duration and the comfort of life.

Some of the fundamentals of the art of living are already widely recognized, such as some measure of personal cleanliness, proper disposal of sewage, protection of water and food from contamination, avoidance of contagion, etc. Marked increase in the duration of life has resulted. If we would reduce the losses from the diseases of degeneration, of wear and tear, we must carry the art of living into the field of personal habits, adjustments to environment in family life, business and pleasure—in everything, avoidance of strain.

It is as simple as this: An automobile that is overdriven will not last as long as one that is used with care. Don't step on the gas all the time!

RETINITIS ASSOCIATED WITH NEPHRITIS

Diagnosis

The white spots in renal retinitis are typically large, cotton-wool-like patches, sometimes resembling snowbanks. When small the spots are more numerous and may be arranged in fan shape or star shape about the macula, but even then they are larger than the spots of arteriosclerotic retinitis. The lesions are bilateral, except in the early stages, while arteriosclerotic retinitis is often limited to one eye.

The two grade into each other, because arteriosclerosis is often complicated by nephritis and vice versa. Hemorrhages are common to both forms.

Edema is an important ophthalmoscopic feature. It is present in some degree in most cases. If severe, diagnosis is easy, as both retina and nerve head are affected. In slight degrees, diagnosis is difficult, because it depends on rather indefinite appearances. The microscopic examination of specimens shows that edema is practically always present in the retina in renal retinitis.

Retinal detachment marks an extreme degree of edema.

Retinal edema is probably an important factor in bringing about the star-shaped arrangement of the retinal spots around the macula. The edema produces lines of tension radiating from the macula and the exudate tends to be deposited along these lines. Thus we see the star-shaped arrangement in retinitis of various forms to which edema is common, such as papilledema, obstruction of retinal vessels, syphilitic retinitis, etc. Another and still more important factor in producing the star-shaped distribution of the spots is the radiate arrangement of the supporting fibre of Henle in the retina.

Visual symptoms may be absent if the macula itself is not involved, therefore, they are not helpful in diagnosis.

Incidence

In early stages of nephritis, retinitis is the exception, in very late stages it is the rule, in other words, the incidence depends on the stage at which the examination is made. In certain forms of acute nephritis (due to massive action of toxins) retinal changes are present very early. For example, in trench nephritis, Greenwood found that if the patients were examined

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within an hour or two of their arrival from the trenches fundus lesions were present in over 90%. Also in eclampsia fundus changes appear early.

In chronic nephritis the incidence as given by various observers who have compiled their statistics varies from one-fifth to one-half the cases depending, as I said, on how long before death the examinations are made.

Subsidence

There is a tendency for both hemorrhages and exudates to become absorbed and disappear. Absorption begins at once. A hemorrhage may disappear by absorption in a few weeks. If more massive it may last for months. Exudates go through various stages toward absorption. The fibrin degenerates and becomes hyaline. Fatty degeneration occurs. Phagocytic cells attack the material. Edema subsides. Even white streaks of perivasculitis disappear. At first, new hemorrhages and new white spots appear faster than the old ones absorb. If the general condition improves the retina may clear up. Thus retinal detachment due to edema of this sort gives a far better prognosis than that due to other causes.

There are so many different kinds of nephritis due to the many different etiological agents, most of which are unknown to the chemical pathologists, that classification is unsatisfactory even to the internist and still more to the ophthalmologist.

How then shall we interpret and classify for ourselves and for the internist if he consults us? Some sort of schematic more or less arbitrary classification is necessary for intelligent study. A poor classification is better than none.

The presence of small white spots, hemorrhages, and sclerosed vessels shows that we have arteriosclerotic retinitis. The spots may be arranged in fan shape, but not usually. The hemorrhages are small, flame-shaped, but not always. The blood pressure is high in most cases, especially in younger patients. On the other hand, the presence of large white patches with edema of the disc and of the retina with small, round, or flame-shaped hemorrhages shows the presence of nephritis. If the spots are in fan or star shape around the macula they are not usually so small individually as are the spots in arteriosclerotic retinitis.

Now suppose that we see some characteristic white patches as well as some signs of arteriosclerosis and report nephritis and the internist says, "No albumen, no casts." The explanation is that the nephritis was an acute exacerbation which has subsided. The arteriosclerosis has affected the kidney vessels enough to make that organ vulnerable and a febrile attack (say influenza, or even a common cold) may have caused a passing acute nephritis with enough toxin to produce white patches in the retina.

In general, the large, white, cotton-wool patches are signs of toxemia. The small, rather scanty, white dots are signs of vascular disease. We may picture to ourselves in a schematic way that some toxic substances acting in *small doses, but long continued,* bring about hypertension of the arteries, later, sclerosis of the arteries, later diseases of any organ where the sclerosis has reached a stage interfering with the blood supply sufficiently to cause local lesions and symptoms and local increased susceptibility to disease (kidney, brain, heart, retina). Toxic substances in *massive* doses probably of different origin and nature produce acute rise of arterial tension, acute nephritis, acute edema and exudation, and hemorrhage in the retina. Combinations of the two types cause all degrees of intermediate gradations.

With this as a basis we can go on to classify hypertension into mild benign, severe benign, malignant, the chief considerations being (a) the height of the diastolic blood pressure (under 115 in mild, over 115 in severe, over 130 in malignant). (b) Extent of arteriosclerosis: mild, none demonstrable in brain, heart or kidneys and mild in retina; severe: some involvement of heart, brain or kidney, marked retinal arteriosclerosis, with hemorrhages and spots; malignant: add to these edema of optic nerve—and (c) the age and prognosis: age, younger in malignant; prognosis, good in mild type

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with suitable treatment, rather bad in severe type depending on extent and stage of arteriosclerosis of heart, kidney and brain; very grave in malignant type.

We can classify the renal cases as our knowledge increases, but at present I have no scheme to offer only the toxic types with abundant cotton-wool patches and edema with a few hemorrhages, the vascular type with rather scanty small dots which may or may not form a star figure and are associated with sclerosis of the vessels.

Wagener has shown (American Journal of Ophthalmology, April, 1924; Vol. 7, p. 272) that renal function is only exceptionally lowered in simple arteriosclerosis of the retina (no white spots, only vascular changes and hemorrhages), that renal function is sufficiently affected to show some slowing of excretion of phenolsulphonephthalein in arteriosclerotic retinitis (white spots as well as hemorrhages and sclerosis of vessels), that in retinitis of hypertension plus nephritis (includes edema) there is temporary renal insufficiency with capacity to return to normal, while in retinitis of nephritic type (includes white patches) there is progressive renal insufficiency with chronic glomerulonephritis.

Conclusions

The retinal changes in cardiovascular-renal diseases may be classified into:

(1) Those where vascular disease dominates the picture; this includes
(a) arteriosclerosis (sclerosis of retinal vessels, hemorrhages);
(b) arteriosclerotic retinitis (the same as (a)), plus white spots.

(2) Those where a toxemia is the dominant factor; these show edema of retina and optic nerve; hemorrhages, especially the small, round, deep-seated ones, but also the striated, superficial ones; exudates in cotton-wool patches and in small spots, often arranged in star or fan-shape around the macula.

(3) Those where both (1) and (2) are much in evidence.

It is not possible to make a diagnosis of the condition of the kidneys from an examination of the retina. The ophthalmoscopic findings will disclose whether vascular disease is an important factor in a given case. If cotton-wool patches are present with edema and perhaps some hemorrhages, we can say that a toxic factor is at work; but we cannot say what the toxin is or that a definite type of nephritis (for example, chronic glomerular nephritis) is present. Various writers have tried to draw conclusions as to renal conditions from fundus appearances. The old name "albuminuric retinitis" is based on the frequency with which albumin is found in the urine in some cases of retinitis. But in seventeen cases reported by Wagener (American Journal of Ophthalmology, April, 1924) where the fundus showed the changes of retinitis with nephritis (hemorrhages, edema, cotton-wool patches, and whitish spots often arranged in star- or fan-shape around the macula), the clinical study of the cases resulted in classification of only ten as nephritis and of seven as hypertension.

The early detection of arteriosclerosis may be made of great benefit to the patient, provided it results in such modification of his habits and mode of life as will tend to arrest the progress of a disease which in its early stages causes little or no reduction in efficiency but is bound to progress if disregarded. Since the early symptoms are negligible, appeal must be made to the patient's intelligence, and his cooperation secured. This calls for the highest type of medical skill and tact. Such patients should no more be left to decide for themselves than children should be permitted to decide whether to go to school or what to eat or wear.

In referring cases with retinal changes to an internist, it is well to make clear to him whether the retina shows vascular lesions—arteriosclerosis, with nephritis possible but not probable; or toxic lesions, with nephritis probable but not certain; suggesting in the former that, while you do not expect him to find any serious disease, you hope he may give the patient such advice and

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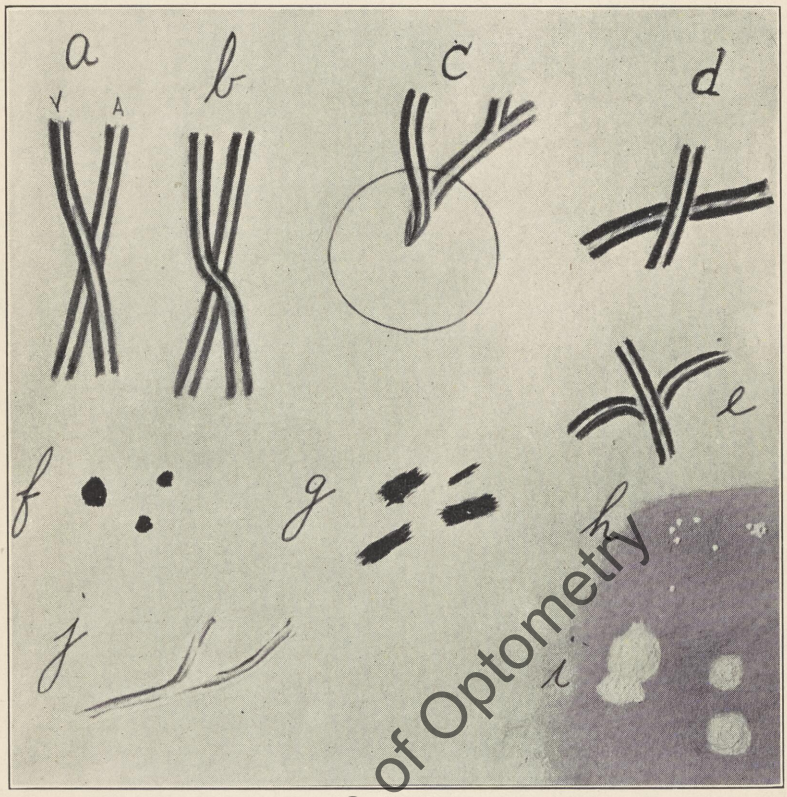

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guidance as will forestall or postpone serious disease, readjusting the patient's life to his capacity to carry on without strain. Otherwise he may simply test the urine for albumin; and if it is negative, advise the patient that it was a false alarm, that everything is satisfactory, and that no treatment is needed. To guide and reform the habits of an intelligent and sensible person should be a simple matter for any general practitioner. The real test of the doctor's superior skill and competence is in the number of foolish, self-indulgent patients that he can manage to reform without making them valetudinarians or worriers.

In order to concentrate the attention on the details which decide the diagnosis, the illustration shows only isolated features of the fundus picture, viz.:

(a) Normal oblique crossing of a vein over an artery;

(b) The change in such an oblique crossing brought about by sclerosis of the artery—rectangular crossing;

(c) Wall of artery visible where it overlies a vein on the disc, often seen in normal eyes;

(d) Visibility of arterial wall, smaller branch not on disc. This is not normal, but shows sclerosis. The wall of the artery obscures the vein which is not visible right up to the dark blood column, as in normal conditions (a);

(e) Vein deflected by overlying artery, which shows sclerosis of its walls obscuring the vein as in (d). Deflection of a vein which curves around an artery is not always due to sclerosis, but may be seen in normal eyes. It is suggestive, however, and if associated, as in this case, with opacity along its border, this hiding of the vein over a space wider than the blood stream is definite evidence of arteriosclerosis;

(f) Hemorrhages approximately round, located in deeper layers of retina. Blood has escaped from very small branches;

(g) Hemorrhages striated or flame-shaped located in nerve-fibre layer (superficial often overlie vessels). Blood has escaped from larger branches, all of which lie in this layer;

(h) Tiny white dots, usually discrete, occasionally confluent. Arteriosclerotic retinitis;

(i) Cotton-wool or snow-bank patches. Larger areas of exudate found in nephritis;

(j) Periarteritis producing "silver wire arteries" or "pipe-stem sheathing."

Dr. Weeks, Portland:

Dr. Lancaster has described the disturbances to which he refers so well and so thoroughly that there is little left for me to add. In regard to the appearance of white spots in the retina, their shape will, as a rule, clearly indicate their location. Wherever they occur in the nerve fiber layer they are more or less spread out. When deep in the retina they are sharply defined.

There is a form of white patch, associated with arteriosclerosis which Dr. Lancaster has not mentioned. I refer to the wreath of white exudation that is present in the so-called retinitis circinata. This exudation comes on in advanced life and extends in an arc above and below, in a horizontal direction, temporally. That portion of the retina within the arc is always disturbed, minute hemorrhages and small patches of exudation appearing. Vision is always reduced. The exudation occurs principally in the nerve fiber layer, it persists for a long period of time, but, in some cases, disappears entirely, accompanied by improvement in vision. The speaker has observed one case from its inception, in which the exudation disappeared entirely.

Dr. Patton, Omaha:

I have nothing to add, except to express my appreciation of the excellent presentation of this subject and emphasize what the author and Dr. Weeks have so ably presented.

Occasionally a patient will come to us for the extraction of a senile

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cataract. Even though the projection is fair, if we find on examining the uninvolved eye that there is a degeneration of the central retina which we have learned to associate with senile changs, we are justified in giving a guarded prognosis. Central senile changes are usually bilateral and while not necessarily a contra-indication, the patient should be warned that post-operative vision may be considerably less than would ordinarily be expected with a normal retina

Dr. Greenwood, Boston:

I wanted to take this opportunity again to thank you and to congratulate you on the meeting you have had here and the excellent papers that have been brought forward by your members.

There are one or two things regarding Dr. Lancaster's paper. First, the co-operation which Dr. Lancaster suggests between the ophthalmologist and the family doctor. We often see the case first and thus we can all be a help to the family doctor by sending him a report of our findings.

Dr. C. A. Veasey, Spokane:

The above discussion of Dr. Lancaster is very valuable and practical. We have had the opportunity to study a great many of these fundus cases and have long since concluded that much more dependence can be placed in a study of the blood chemistry than in that of the urine. In many cases, in which an examination of the urine fails to show even a trace of albumin or tube casts, a blood chemistry may reveal a very serious condition of the kidneys and cardio-vascular system. One case which we have followed for a very long time, who has a chronic nephritis following scarlet fever, has had three attacks of uremic coma, and yet most of the time the urinary examination reveals no albumen and no, or very few, tube casts. A blood chemistry, however, reveals a high non-protein nitrogen and at times a moderately high creatinin. Because of these experiences we have come to depend greatly upon the results of our blood chemistry examination, whether or not albumin and tube casts be found in any examination of the urine.

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BUSINESS MEETING

MONDAY, JUNE 6TH, 1927

The annual business meeting of the Pacific Coast Oto-Ophthalmological Society was called to order in the Davenport Hotel by Dr. Carroll Smith, and the following report of the Executive Committee was read:

The meeting of the Executive Committee was called to order by the President, Dr. Carroll Smith, at the Davenport Hotel, Sunday, June 5th, 1927.

The following men were present: Dr. Smith, Dr. Pischel, Dr. Mellinger, Dr. Cunningham, Dr. Hoffman.

The audit of the certified public accountant was inspected and found to be satisfactory.

The matter of defraying the expenses of the secretary was brought up by Dr. Smith. Dr. Hoffman reminded the committee that at the last executive committee meeting, on the instance of Dr. Kiehle, the secretary was voted $20.00 a month for expense between meetings. Dr. Smith did not think this sufficient and after considerable discussion, Dr. Cunningham moved, Dr. Pischel seconded that the secretary receive $300.00 a year. Motion passed.

The secretary read the letters of resignation of the following:

Dr. John F. Beaumont,
Dr. S. J. Torney,
Dr. Vard Hulen,
Dr. H. S. McGavern.

Above resignations were accepted.

Considerable discussion of the Los Angeles-Pasadena situation, where extreme initiation fee makes it almost impossible for the younger men to go into the local County Society, and the executive committee recommended, following the discussion, that the secretary write the Los Angeles County Medical Society inquiring into the status of the situation of the fee. Using this letter as a basis, write a second letter to the Los Angeles County Society covering our attitude in the matter, explaining the reason for our attitude and the possibility of our writing to the A. M. A. in regard to this, in case the Los Angeles Society does not relieve the situation.

The following applicants were recommended for membership in the Society:

Eugene Christenson	Vard Cooper	Manford Waltz
H. L. Goss	Samuel Durr	Grace Young
L. L. Henninger	Frederick Harrison	Harold Van Cott
Cyril Lundvick	H. W. Hunsaker	Montie D. Comer
P. C. Means	James Mathews	W. A. Briggs
Merton Price	Paul Neely	Dwight Miller
Frank Slopansky	Randolph Sharpsteen	John A. Donovan
W. H. Wimp		

The following names were accepted for membership subject to their passing either the Board on Ophthalmology or the Board on Laryngology:

Albert Aland	A. W. Howe
L. B. Auerbach	F. I. Putnam
B. R. Washburn	J. F. Gibson
W. Le Roy Smith	

Letters of invitation from Salt Lake City, Yosemite National Park, and Santa Barbara were read and considerable discussion of the matter followed.

Dr. Pischel moved, Dr. Cunningham seconded, that we accept the Santa Barbara invitation.

Dr. Pischel nominated, Dr. Cunningham seconded, Dr. Mellinger for president. Dr. Hoffman moved, Dr. Smith seconded, Dr. Hampton for First Vice-President. Dr. Cunningham moved, Dr. Mellinger seconded the name of Dr. Wanamaker for Second Vice-President.

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Dr. Cunningham moved and Dr. Pischel seconded that Dr. Hoffman be re-elected Secretary-Treasurer. All motions passed.

Dr. Fenton suggested that the following men were eligible for honorary membership at this time:

Dr. H. W. Loeb, St. Louis;
Dr. Frank Spencer, Boulder, Colo.;
Dr. Wm. Wilder, Chicago;
Dr. Walter Lancaster, Boston;
Dr. R. Clyde Lynch, New Orleans;
Dr. Ross Skillern, Philadelphia;
Dr. W. P. Wherry, Omaha, Neb.;
Dr. Allen Greenwood, Boston;
Dr. James B. Patton, Omaha;
Dr. Thomas Halstead, Syracuse.

As there was no further business the meeting adjourned.

Dr. Carroll Smith asked the pleasure of the Society as to the report of the Executive Committee. A member asked from the floor if the Society should vote on the report as a whole or take up each item separately. President Carroll Smith suggested that each item had best be discussed and voted on separately.

Dr. Seely moved, Dr. Jordan seconded, that the recommendations of the executive committee in regard to a $300.00 compensation for the secretary be adopted. Motion passed.

It was moved by Dr. Cameron, seconded by Dr. ———— that the candidates for membership be elected as per the recommendation of the executive committee. Motion passed.

It was moved by Dr. Hyde, seconded by Dr. Jordan that the Society meet in Santa Barbara next year. Passed.

Dr. Seely moved, Drs. Hyde and Jordan seconded, that the officers as recommended by the executive committee be elected. Passed.

The attached report from the Committee on Legislation was read:

Mr. Chairman and Members of the Pacific Coast Oto-Ophthalmological Society:

The following is a report of your Committee on Public Health and Legislation for the year of Nineteen Hundred and Twenty-seven. The Chairman has been in touch with the different members of the committee representing their respective States and Provinces, either personally or by communication, and through their unfailing energy and cooperation, we have been able to summarize the Legislation pertaining to Medicine in general and our specialty in particular, as follows:

John J. McLoone of Phoenix, member of the Committee for Arizona, reports that a bill, drafted in keeping with the National model Lye and Caustic Bill, was introduced in his State the latter part of January. It was reported upon favorably by the Senate. There were some objections from local grocers. These were met, however, by an amendment which in no way affected the value of the bill. It was again referred to the Committee on Public Health Legislation where it was favorably reported and was expected to pass both Houses and be returned to the Governor for his signature. A certain bill, which would have enabled the osteopaths to use the hospitals of the State of Arizona, was killed. The Basic Science Practice Act, which is a great step forward toward putting the medical practice on a higher plane, was introduced into the Senate. Unfortunately, however, it did not pass.

F. W. Brydone-Jack, member of the Committee for the Province of British Columbia, because of his absence abroad, was unable to be present at our conference. He has, however, carefully reviewed the legislation of his Province and states that there is no legislation covering the branding and sale of containers of lye and other caustic cleansing agents, but that an Act, Schedule B, Sections 24, 25, 27, 28 and 34, covers Oxalic Acid, Cresol and its preparations, and other groups of poisonous drugs. The ophthalmia neonatorum law seems to be adequate and well enforced.

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George H. Kress, member of the Committee for California, has reported in detail the progress of legislation in his State in regard to three major measures of particular interest to Eye, Ear, Nose and Throat specialists. Two vicious bills which were introduced failed of passage. The first would have given the Optometric Board, and more particularly, certain designing optometrists who had licenses from the Board, a marked advantage over the oculists. The second bill would have made it a misdemeanor for any physician to fail to report a minor with defective hearing. In regard to Lye Legislation, measures are on foot in California whereby the essential elements in the National Lye Legislation will be made applicable to lye and similar caustics made and sold in the State of California.

C. W. Pond, member of the Committee for Idaho, reports that they have been successful in getting a man appointed to the Industrial Board who is in sympathy with the regular medical profession. Several bills pertaining to the regulating of hospitals as affected by the Industrial Accident Law have been defeated or satisfactorily amended. The medical profession of the State of Idaho scored a great victory in the defeat of the so-called Osteopathic Surgery Bill. A bill was introduced covering uniform legislation on the sale and branding of containers of lye and other caustic cleansing agents but for lack of time was not passed, but undoubtedly will be enacted at the next session. The State of Idaho has a law governing the prophylaxis and treatment of ophthalmia neonatorum, which is considered to be one of the best in the United States.

John A. Fuller, member of the Committee for the State of Nevada, reports that Nevada has passed a bill similar to the National Bill regulating the branding and sale of caustics, acids and other similar preparations for household and other indiscriminate use. Nevada also has a statute which requires a doctor or a nurse to put a solution of silver nitrate of one per cent, or a stronger solution of one of the organic derivatives, into the eyes of the new-born. This law has been enforced to the effect that a report of such treatment must accompany the birth certificate. The Nevada Legislature also defeated the Sheppard-Tanner Bill, a bill which has been fought by the local and State Medical Societies since its inception.

C. C. Albright, member of the Committee for Montana, stated in a personal communication, that the medical legislation in his State was well in hand. He also stated that, personally, he was in favor of having a more stringent law drafted relative to the sale and handling of high explosives and detonation caps, as he has encountered many cases of serious eye injuries in children, the result of the negligence and failure of miners and blasters to properly safeguard the interest of these little ones.

J. J. Emmens, member of the Committee for Oregon, was well satisfied with the condition in his State and feels that the medical profession has the Legislature well under control, as the profession is well organized and represented by the Public Health League of the State of Oregon.

The State of Washington is fortunate in having a Public Health League which has functioned since 1920 in the interests of the medical profession. The outstanding constructive achievements of the League, so far as legislative matters are concerned, include the License Revocation Bill and the Basic Science Bill. The License Revocation Bill provides machinery for the revocation of "diploma mill" licenses and establishes quick procedure for the same.

The Basic Science Bill provides that any applicant for license to practice the healing art must first qualify by passing tests in the so-called fundamental subjects of anatomy, physiology, chemistry, pathology and hygiene. The committee to conduct this examination is recruited from the faculties of the University of Washington and the Washington State College. This bill is a very direct blow against quackery, insufficient training, insecure education, trickery in examination and "diploma mills."

A number of proposals and bills have been defeated. Those noted below are shown as an indication of the continual attack being made against scientific medicine and the public health:

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1. Bill to license Abrams healers, giving this cult medical recognition.
2. Bill to establish the "open door for hospitals whereby osteopaths, chiropractors, sanipractors and Christian Scientists are given the right and power to enter hospitals for practice.
3. Bill requiring all medical practitioners to write their prescriptions in English.

The above bills of this State, and similar bills of other States and Provinces, make apparent the need for constant watchfulness and concerted action on the part of the medical profession.

Respectfully submitted,

E. F. Chase, *Chairman*	J. J. Emmens	J. J. McLoone
C. C. Albright	J. A. Fuller	H. G. Merrill
F. W. Brydone-Jack	Geo. H. Kress	C. W. Pond

As there were no deaths, no report from the Necrology Committee was read.

The secretary-treasurer submitted the following report:

Balance December 31, 1926	$3,130.57
Dues and Initiation Fees	1,450.00
Total	4,581.37
Paid out by Checks or Bank Exchange	841.64
Balance June 1, 1927	$3,739.73

Dr. Cameron said he was a modest man from a modest town, but that Tacoma offers every advantage for a meeting, and invited the Society to meet there in 1929.

Dr. Stillson moved that the Society express their appreciation to the members of the Examining Boards and the President and Secretary of the Society for their efforts in making the meeting a success.

Dr. Seely moved that we send a letter of sympathy to Dr. Lynch in regard to the Mississippi flood condition and thank him for sending the motion picture. Dr. Kiehle seconded this motion and amended it to the effect that we send Dr. Lynch $500.00 for the Flood Relief Fund. Dr. Seely accepted the amendment.

Dr. Dickson moved that we increase the amount to $1,000.00, but the motion was lost because of want of second.

Dr. Jordan spoke of the hospitality of the Spokane men and moved a rising vote of thanks.

Dr. Mellinger then offered a few words as to the qualifications of Santa Barbara as a meeting place.

Dr. Donovan, chairman of the Section on Ophthalmology A. M. A., urged the members to attend the Section of Ophthalmology at the Minneapolis meeting next year.

Being no further business, the meeting adjourned.

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CONSTITUTION AND BY-LAWS

ARTICLE I

NAME

The name and title of this Association shall be the Pacific Coast Oto-Ophthalmological Society.

ARTICLE II

OBJECT

The object of this Society shall be: the cultivation and advancement of Medical Sciences, in general, especially the branches which pertain to diseases of the Eye, Ear, Nose and Throat; to co-operate with the various County and State Medical Associations for the extension of medical knowledge, and the advancement of medical education, and to the enacting and enforcing of just medical laws; to the promotion of friendly intercourse among its members and to the guarding and fostering of their material interest; to the enlightenment and direction of public opinion in regards to the great problems of medicine and surgery, so that the profession shall become more capable and honorable within itself, and more useful to the public in prevention and cure of disease, and in prolonging and adding comfort to life; to purchase, receive, acquire, own and hold all such personal property as may be necessary or proper, or convenient, for carrying out the purposes of this organization.

ARTICLE III

MEMBERSHIP

Section 1. The Society shall consist of Active Members, Honorary Members and Guests.

Section 2. Active Members—Every respectable and legally qualified physician residing in the states of California, Oregon, Washington, Idaho, Montana, Arizona, Nevada, Utah and British Columbia, whose practice is limited to the Eye, Ear, Nose and Throat, and who does not support, practice or claim to practice sectarian medicine; who has a membership in good standing in his local County or District Society, and also a membership in good standing in his local Eye, Ear, Nose and Throat Section or Society where such Section or Society exists; who has had special training in one or more of the aforementioned specialties, to the equivalent of one year's work in Clinics or Post Graduate Schools, in which these specialties are taught, shall be entitled to active membership, by complying with the rules and regulations of the Society as hereafter set forth.

Section 3. Applications for membership shall be made in such form as shall be provided, accompanied by membership fee, in accordance with Article VIII. All elections shall be by ballot and four-fifths vote of all members present shall be necessary for election, applications having been approved by the Executive Committee.

Section 4. Applications from rejected candidates shall not be received within one year of such rejection.

Section 5. All active members shall be equally privileged to attend all meetings, and take part in all proceedings, and shall be eligible to any office or honor within the gift of the Society so long as they conform to these By-Laws, including the payment of dues to the Society, provided that no member under sentence of suspension or expulsion shall take part in any proceedings of the Society or be permitted to resign from the Society or be eligible to any office in the Society until relieved of such disability.

Section 6. All charges against a member shall be made in writing to the Executive Committee and the same shall be investigated at their discretion, and if found to be of sufficient moment, after the accused has been given the privilege of a hearing, shall be reported to the Society for action.

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A member who has been found guilty of a criminal offense, or of gross misconduct, either as a physician or as a citizen, or whose license to practice medicine in his State or District has been revoked by the State Board of Examiners, or who has committed any act which may be derogatory to the medical profession, or who shall refuse or neglect to obey the regulations of this Society, or who has violated any of the provisions of these By-Laws, shall be liable to censure, suspension or expulsion, which censure, suspension or expulsion shall require two-thirds affirmative vote of the members present at a regular meeting.

Written notice of charges preferred must be given to the accused ten (10) days in advance of such meeting, and opportunity of accused to be heard in his own defense before a vote of the Society is taken on his censure, suspension or expulsion.

Section 7. Any physician who shall procure, for profit, a patent for a remedy or an instrument of surgery, or who sells, or who is interested in the sale of patented remedies, or nostrums, or shall give a certificate in favor of a patented or a proprietary remedy, or patented instrument, or who shall enter into agreement to receive pecuniary compensation, or patronage for sending prescriptions to any apothecary, or for sending orders to an instrument manufacturer or retailer or optical company, shall be liable to censure, suspension, or expulsion in accordance with Article III Section 6 (it being the intent of this clause to prohibit the acceptance of rebates or commissions, but not to prevent a physician from selling to his patients drugs, appliances or spectacles or eye glasses, when in his opinion the patient's interest may be protected by so doing).

Section 8. The Code of Ethics of the A. M. A. is hereby adopted by this Society and shall be considered a part of the By-Laws of this Society.

Section 9. Distinguished physicians and others who are not members of this Society may be elected Honorary Members by a four-fifths vote of those present, but they shall not be entitled to privileges of active members. The lists of Honorary Members shall be published in connection with the membership rolls of the Society.

Section 10. The Executive Committee may invite Distinguished Physicians and Members of Allied Sciences as Guests to read papers at any regular or special session of the Society. Such Guests shall have the privileges of participating in all scientific subjects which come before the sessions to which they have been invited.

ARTICLE IV

Meetings

Section 1. One meeting shall be held annually at such place as may be designated by a majority vote of members present at any regular meeting; the meeting place shall be selected one year in advance; the time of the annual meeting shall be fixed by the Executive Committee, which will be best suited to the climate and other facilities of the place selected for the meeting. Twenty members shall constitute a quorum.

Section 2. Special meetings may be called by the Executive Committee

ARTICLE V

Officers

Section 1. The officers of the Society shall consist of a President, First and Second Vice-Presidents, Secretary-Treasurer and an Executive Committee of Five Members, which shall consist of the President and Vice-President, Secretary-Treasurer, and the two past Presidents. The President shall be Chairman of the Committee.

Section 2. The President, Vice-President and Secretary-Treasurer shall be elected at the annual meeting. The Executive Committee shall nominate a full ticket from the active members of the Society. The election of officers shall be the first order of business, at the last general assembly of the annual

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sessions. Nominations to any elective office may be made in the general assembly. A majority of all votes cast is required for election.

Section 3. The President shall preside at all meetings of the Society and Executive Committee, and have general supervision of all the affairs of the Society and enforce the rules and regulations thereof, and perform and discharge all the duties usually performed by a President of a Society.

Section 4. The First Vice-President shall be a member of the Executive Committee, shall assist the President in the performance of his duties, shall preside in his absence and upon his death, resignation or removal from the district, shall succeed to the Presidency.

Section 5. The Secretary-Treasurer shall be a member of the Executive Committee. He shall record the minutes of the Society and Executive Committee meetings. He shall receive and keep account of all dues and moneys belonging to the Society and shall pay out the same only upon the written order of the President. He shall give bond for the trust reposed in him and shall annually subject his accounts to examination by a certified accountant, the expense of both of which shall be paid from the funds of the Society.

He shall notify each member of the Society as to the time and place of each meeting, and, whenever possible, give the program for the meeting. He shall make and keep a list of the members of the Society in good standing, noting of each his correct name, address, place and date of birth, place and date of graduation, and date of license to practice medicine in this district.

Section 6. The Executive Committee shall be the governing body of the Society and shall have full power and control of all the property of the Society, with the authority to sell and dispose of the same or any part thereof. It shall have the power to make such rules and regulations for the comfort and success of the Society as it may deem proper.

It shall have the power to remit the entrance fee of any member or his dues for a period of not more than one year following the date of such motion.

It shall have the power to extend the courtesies of the Society to non-resident visitors, to arrange for affiliation of this Society with other Societies and to decide all questions not governed and determined by the By-Laws. No funds shall be disbursed without the vote of the Executive Committee.

The Executive Committee shall meet at least once a year, such meetings to be determined by itself.

The Executive Committee shall examine and report on the qualifications of applicants for membership, subjecting each applicant to such examination as it may deem necessary, it shall investigate charges preferred against a member and report its conclusions and recommendations to the Society.

ARTICLE XI

Committees

Section 1. The incoming President, on taking his chair, shall appoint standing committees as follows:

A Committee on Program and Scientific Work.

A Committee on Public Health and Legislation.

Section 2. The Committee on Program and Scientific Work shall consist of five members. It shall be its duty to promote scientific and social functions of the Society by arranging attractive programs for each meeting.

ARTICLE VII

Funds and Expenses

Section 1. The entrance fee, which must accompany the application, shall be ..... dollars. The entrance fee shall be returned if the applicant is not accepted.

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lowing, the member shall be held as suspended from the Society, and his name shall be placed on the list of non-affiliating physicians, and shall remain so until such disability is removed.

ARTICLE VIII

Order of Business

The order of business shall be as follows:

1. Call to order by the President.
2. Reading of Minutes of last meeting.
3. Clinical cases and Pathological specimens.
4. Papers and discussions.
5. Unfinished business.
6. New business.
7. Reports of Committees.
8. Announcements.
9. Adjournment.

ARTICLE IX

Rules of Order

The deliberations of this Society will be governed by parliamentary usage as contained in Roberts' Rules of Order, unless otherwise determined by vote. Papers by members shall be limited to twenty minutes time, and the discussion to five minutes.

ARTICLE X

Amendments

This Society may amend any article by a two-thirds vote of its members present at a regular meeting and notice shall have been sent by mail to each member ten days in advance of the meeting at which final action is to be taken.

ARTICLE XI.

Application for Membership

Beginning January 1, 1927, no application for membership shall be received except from such individuals as have been certified to by the American Board for Ophthalmic Examinations or the National Board for Otolaryngology, with the exception of those physicians in good standing in practice thirty years or more.

By-Laws

Annual Dues and Entrance Fee

Section 1. The annual dues shall be Five Dollars for all active members, payable on January First. The entrace fee, which must accompany the application, shall be.....dollars. The entrance fee shall be returned if the applicant is not accepted.

Annual Banquet

Section 2. The expense of the annual banquet will be met by a plate charge for each one participating with the exception of honorary guests.

Expense of Meeting

Section 3. The expense of the annual meeting shall be met by a five dollar ($5.00) registration fee charged each member attending the annual meeting.

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MEMBERS OF THE PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

HONORARY MEMBERS

Beck, Joseph C.....Chicago, Ill.
Benedict, W. L.....Rochester, Minn.
Burch, Frank E.....St. Paul, Minn.
Carmody, Thomas E.....Denver, Colo.
Davis, A. E.....New York City
Elliot, Col. R. H.....London, Eng.
Greenwood, Allen.....Boston, Mass.
Jackson, Edward.....Denver, Colo.
Parker, Walter R.....Detroit, Mich.
Fischer, Martin.....Cincinnati, O.
Halstead, Thomas.....Syracuse, N. Y.
Hawley, C. W.....Chicago, Ill.
Lancaster, Walter.....Boston, Mass.
Loeb, H. W.....St. Louis, Mo.
Lynch, R. Clyde.....New Orleans, La.
Mosher, Harris P.....Boston, Mass.
Patton, James B.....Omaha, Nebr.
Shurly, Burt R.....Detroit, Mich.
Skillern, Ross.....Philadelphia, Pa.
Spencer, Frank.....Boulder, Colo.
Parsons, Sir John.....London, Eng.
Weeks, John E.....Portland, Ore.
Reik, H. O.....Baltimore, Md.
Swift, Geo. W.....Seattle, Wash.
Wherry, W. P.....Omaha, Nebr.
Wilder, William.....Chicago, Ill.
Wilson, J. Gordon.....Chicago, Ill.
Wood, Wm. L.....Oracle, Ariz.

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Barkan, Otto.....516 Sutter St., San Francisco, Calif.
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